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# Endangerment Assessment Report Cold Creek/LeMoyne Site, Mobile County, Alabama

Prepared for:

Akzo Chemicals, Inc. Chicago, IL

ICI Americas Wilmington, DE

May 1988



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#### Introduction

The Endangerment Assessment for the Cold Creek/LeMoyne site is based on existing data from the Remedial Investigation and other information provided to ERT by Stauffer Chemical Company. The development of this Endangerment Assessment (EA) is consistent with the requirements of the National Contingency Plan (NCP), and conforms to EPA guidance regarding EAs at CERCLA sites.

#### Site Description and History

Stauffer Chemical Company previously owned and operated a multi-product inorganic chemical manufacturing plant at LeMoyne, Alabama and an agricultural chemical facility at the adjacent Cold Creek site. The LeMoyne plant, purchased by Akzo Chemie America, Inc. (now Akzo Chemicals Inc.) in 1987, has been in operation since 1953. The Cold Creek plant has been in operation since 1966 and is currently owned by ICI Americas, Inc.

Halby Chemical Company (later part of Witco, Inc.) also operated a small facility for a time on a leased section of the LeMoyne property.

Until 1973, industrial wastes from these operations were disposed in unlined disposal areas and, in the case of wastewater, to unlined ponds or, after treatment, by discharge to Cold Creek swamp. Presumably as a result of these practices, a ground-water contamination problem developed. This was recognized in the early 1970's, and many improvements and waste-handling modifications were made, including the installation of a ground-water intercept and treatment system.

In 1982, an assessment of the plant sites was made by the Alabama Department of Public Health in response to submissions made by Stauffer to the House Committee on Interstate Commerce

("the Eckhardt Survey"). At the request of the Alabama
Department of Public Health, monitoring wells were installed
around the three closed landfills. In spite of the previously
identified ground-water problems already under remediation,
data primarily from these monitoring wells were held by the
Federal Environmental Protection Agency (EPA) to be the basis
for inclusion of these facilities on the National Priorities
List (NPL), which ranks hazardous waste disposal sites under
provisions of the Comprehensive Environmental Response,
Compensation and Liability Act of 1980 (CERCLA), commonly known
as "Superfund".

#### Extent of Contamination

To properly assess the potential public health and environmental impacts from the Cold Creek/LeMoyne site, a characterization of the potential areas of contamination must be made. These include the nine ponds or lagoons, four of which are still active, the three closed landfills, and portions of the two swamps (Cold Creek and LeMoyne).

Based on the frequency of detection, the concentrations detected, and the toxicological properties of the contaminants which have been found at the site, the following compounds were selected as "representative" compounds. These are:

- carbon tetrachloride;
- carbon disulfide;
- cyanide;
- mercury;
- 6 thiocarbamates (including EPTC, butylate, cycloate, molinate, pebulate, and vernolate); and
- thiocyanate.

#### Exposure Assessment

The Cold Creek/LeMoyne site is located in an industrial area and is surrounded by several other large chemical production companies. Fewer than 10 residences are located within one mile of the site, and none of them are downgradient of the contaminated ground water at the site. The nearest population centers include Mt. Vernon (with a population of 1,038), which is located about 8 miles north of the site, and Creola (population of 673), which is located about 5 miles to the south (U.S. Department of Commerce, 1981).

The majority of the chemical plants as well as the local communities in the area obtain water from the water-table aquifer. The Cold Creek facility has one drinking-water well and one backup well, and the LeMoyne facility has two drinking-water wells. The CNA facility to the south has one drinking-water well and a backup well. These wells were sampled during the RI, and no contaminants were detected.

Both Cold Creek swamp and LeMoyne swamp represent the most important environmental receptors at the site. These swamps currently support a diverse variety of plants and animals, including the alligator, which is currently listed as a threatened species. The Mobile River, which forms the eastern boundary of the site, is also a potential environmental receptor.

After identifying the potential receptors and the contaminants to which they may be exposed, it is necessary to determine the ways in which they may be exposed and the frequency and magnitude of the potential exposure. Human exposure to the contaminants identified at the Cold Creek/LeMoyne site can potentially occur directly through air, water or solid media (soils, sediments or sludges) or indirectly through the food chain; however, the most likely exposure pathways are as follows:

- Incidental ingestion of contaminated swamp sediments;
- dermal contact with contaminated swamp sediments;
- ingestion of contaminated biota; and,
- ingestion of contaminated ground water.

Exposure to contaminated swamp sediments and via ingestion of fish is probably infrequent because the site is located in an industrial area and people generally do not spend much time in wetland areas unless they are bird watchers or are on other kinds of nature walks. In addition, shoes and other articles of clothing will help to protect anyone from direct contact with the swamp sediments. Ingestion of contaminated fish is also expected to be infrequent because it is unlikely that anyone fishes in Cold Creek swamp, given its proximity to industrial property, its restricted access, and the small size of the fish in the swamp.

No current risk appears to exist from exposure to contaminated ground water at the site, because none of the contaminants have been detected in any of the drinking-water wells in the immediate vicinity of the site. It is highly unlikely that future exposure to contaminated ground water would occur, because a permit is required for the installation of a potable water well from the state of Alabama, and it is unlikely that a permit would be granted given the documented ground-water contamination at the site.

#### Public Health Risk Characterization

Quantitative risk estimates were conducted for both adult workers and teenagers that could be exposed to contaminants at the Cold Creek/LeMoyne site. Using conservative assumptions, risks were calculated for hypothetical individuals who may come in contact with hazardous compounds via exposure to contaminated swamp sediments and ingestion of contaminated fish and ground water. Two scenarios were developed for each exposure route. The worst-case scenario assumes that an

individual is exposed to the maximum concentration of the compound measured in that media (i.e., sediments or fish), and the realistic case uses average concentrations found at the site. For teenagers, it was assumed that they would be exposed to contaminated swamp sediments 6 times per year for a period of 5 years, and for adult workers it was assumed that they be exposed 12 times per year (once a month) for 30 years. In determining risk from ingestion of contaminated fish, it was assumed that an individual would consume 26 grams of fish per day for a period of 30 years.

Results of the quantitative risk assessment for noncarcinogens show that individuals exposed to contaminants at the Cold Creek/LeMoyne site are not at risk, even if they are exposed to maximum concentrations under current conditions. Even when the risks for each exposure route and each compound are summed, the total Hazard Index (HI) is still less than unity, showing that the estimated doses are less than the threshold doses (i.e., the dose below which no adverse effects are expected to occur). However, care must be taken when interpreting summed HI's, because adding them assumes that their toxicological effects are additive, which may not be true.

The underlying assumption for assessing the risks from suspect or known human carcinogens is that there is no threshold for an adverse health effect. Carcinogenic risk is quantified by multiplying an EPA-derived cancer potency factor by the estimated intake (dose) to calculate cancer risk due to each site-related exposure. Carbon tetrachloride is the only indicator compound at the Cold Creek/LeMoyne site which is considered by the EPA to be a suspect human carcinogen. This compound was only detected in the ground water and in subsurface soil samples from beneath the wastewater treatment ponds. As discussed above, there is no current risk from exposure to contaminated ground water, and therefore, there is no current risk from carbon tetrachloride at this site. However, carcinogenic risk from carbon tetrachloride ranges

from  $3.57 \times 10^{-4}$  to  $5.28 \times 10^{-5}$  should a worker ingest contaminated ground water in the future for a period of thirty years, which is highly unlikely.

#### Environmental Impacts

The most environmentally sensitive portion of the site is believed to be the Cold Creek swamp. Sediment concentrations and fish body burdens of mercury appears to be elevated in this area. Potential exposure routes for aquatic and semi-aquatic organisms include water, sediment and food-chain pathways, with the food-chain exposure route predominating at higher trophic levels. The U.S. Fish and Wildlife Service (USFWS) considers the swamp to be a preferred habitat for the threatened American alligator (Alligator mississippiensis), and the potentially endangered Alabama Red-Bellied turtle (Pseudemys alabamensis).

Current data are not adequate to assess the exposed biota populations or estimate exposure via all pathways and thus to estimate potential risks to these populations. With the data at hand, the potential for adverse affects to sensitive birds and mammals, if these inhabit the wetland, cannot be ruled out.

#### 1. INTRODUCTION

The following draft report is an endangerment assessment for the Cold Creek/LeMoyne site located in Mobile County, Alabama. It was prepared for Stauffer Chemical Company by ERT, a Resource Engineering Company. The evaluation is a risk assessment of the current condition of the site and is intended to determine if endangerment of human health or the environment exists as a result of contamination. As such, it indicates whether a remedial action is needed and how quickly steps must be taken.

Generally, the first step of an endangerment assessment is to identify those compounds which may pose a threat to human health or the environment. These chemicals of concern (or indicator compounds) were identified during the Remedial Investigation (Stauffer, 1987). These are:

Carbon Tetrachloride:

Carbon Disulfide:

Cyanide:

Mercury:

Six Thiocarbamates (including EPTC, butylate, cycloate, molinate, pebulate, and vernolate); and, Thiocyanate.

The first section of this endangerment assessment (Section 2) provides a general overview of the site, including a summary of the site history and the extent of contamination. Section 3 contains a description of the environmental fate and transport of each of the indicator compounds. The toxicity assessment (Section 4) provides a review of the toxicity of each indicator chemical (primarily from the literature supporting standards and criteria) and an estimation of the relationship of quantity of intake (dose) to risk of toxic response. Section 5 identifies exposure points, estimates exposure point concentrations and describes the calculation of expected doses from plausible exposure scenarios.

Section 6. Risk Characterization, is a comparison of expected intake to dose-response estimation in order to quantitate risk for specific site conditions, and Section 7 (Uncertainty Analysis) describes the sources of uncertainty and their effect on the quantification of risk.

#### 2.1 Site Description

Stauffer Chemical Company previously owned and operated two adjacent facilities in Mobile County, Alabama. The LeMoyne plant, purchased by Akzo Chemie America, Inc. in 1987, has been in operation since 1953 and manufactures multi-product inorganic chemicals, including carbon disulfide, carbon tetrachloride, sulfuric acid, chlorine and Crystex (a proprietary sulfur compound). The Cold Creek plant manufactures agricultural herbicides and pesticides, including several thiocarbamates. The Cold Creek plant has been in operation since 1966 and is now owned by ICI Americas, Inc..

In the past, the Halby Chemical Company (HCC) leased a small parcel of land on the western portion of the site. Little is known of this operation, however, other than that waste products and effluents from the facility were held in a pond on the property.

The Cold Creek/LeMoyne site ("the site") occupies 947 acres between the Mobile River and U.S. Highway 43, approximately 20 miles north of Mobile, Alabama (see Figure 2-1). The site is bounded by Virginia Chemicals to the north, Courtaulds North America (CNA), another chemical company, to the south, the Mobile River to the east, and Route 43 to the west. M&T Chemicals is located immediately to the west of Route 43 (Stauffer, 1987).

The site is situated in a predominantly industrial area, but several sparsely populated rural communities are located within a few miles of the site. Maximum relief at the site is on the order of 30 feet. The Cold Creek swamp lies between the plant facilities at the site and the Mobile River, and flows to the northeast and then east to discharge into the river. Surface-water drainage on site is generally toward the swamp or the river, and is governed by a drainage divide between the two. The Mobile River flows to the south and discharges into the Gulf of Mexico.

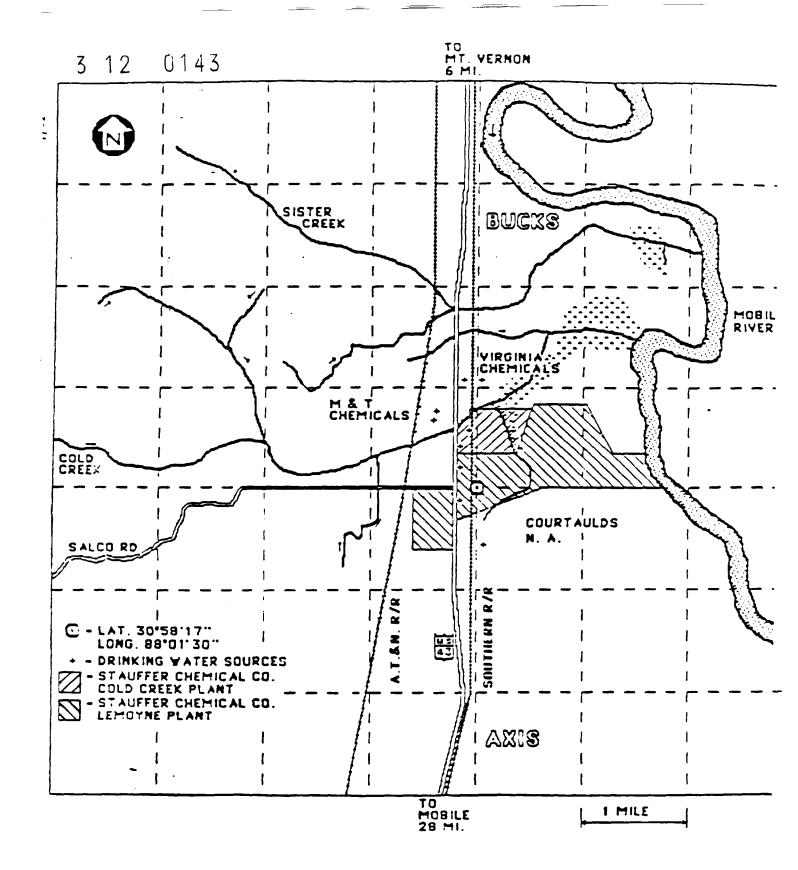


Figure 2-1 Approximate Location of the Cold Creek/LeMoyne Site Mobile Co., Alabama (Modified after Stauffer, 1987)

The majority of the chemical plants as well as the local communities in the area obtain water supplied from the water table aquifer. As shown in Figure 2-2, the Cold Creek facility has one drinking-water well (CC-12) and one backup well (CC-11). The LeMoyne facility has two drinking-water wells (LM7 and LM10). The CNA facility to the south has one drinking-water well (CNA-16) and a backup (CNA-4).

#### 2.2 Site History

#### 2.2.1 Waste Disposal Practices

For approximately 20 years after initiation of operations at the Cold Creek/LeMoyne site, industrial waste products resulting from the Stauffer processes were disposed of in unlined surface disposal sites. From 1965 to 1974, solid waste from the LeMoyne plant was placed in an unlined landfill approximately one mile east of the plant. According to Stauffer (1987), "between 11,000 and 12,000 tons of brine muds were placed in this landfill along with plant refuse, used samples, and some absorption oil."

Two waste disposal sites, referred to as the north and south landfills, are located on the Cold Creek plant property. The exact quantity of material placed in the landfills is not known; however, sludges and solid wastes containing a variety of herbicides and pesticides are thought to have been buried in these two landfills (Stauffer, 1987). The LeMoyne and the two Cold Creek landfills were closed and capped with liners in 1974.

Waste waters from the Stauffer processes were held in unlined lagoons and discharged to the Cold Creek swamp. As shown in Figure 2-2, there are currently five closed or inactive waste water ponds and four active ponds that were investigated during the Remedial Investigations. The four active ponds, LeMoyne LeCreek, Cold Creek LeCreek, LeMoyne acid plant wastewater treatment pond, and the new carbon tetrachloride plant wastewater treatment (WWT) pond, are all

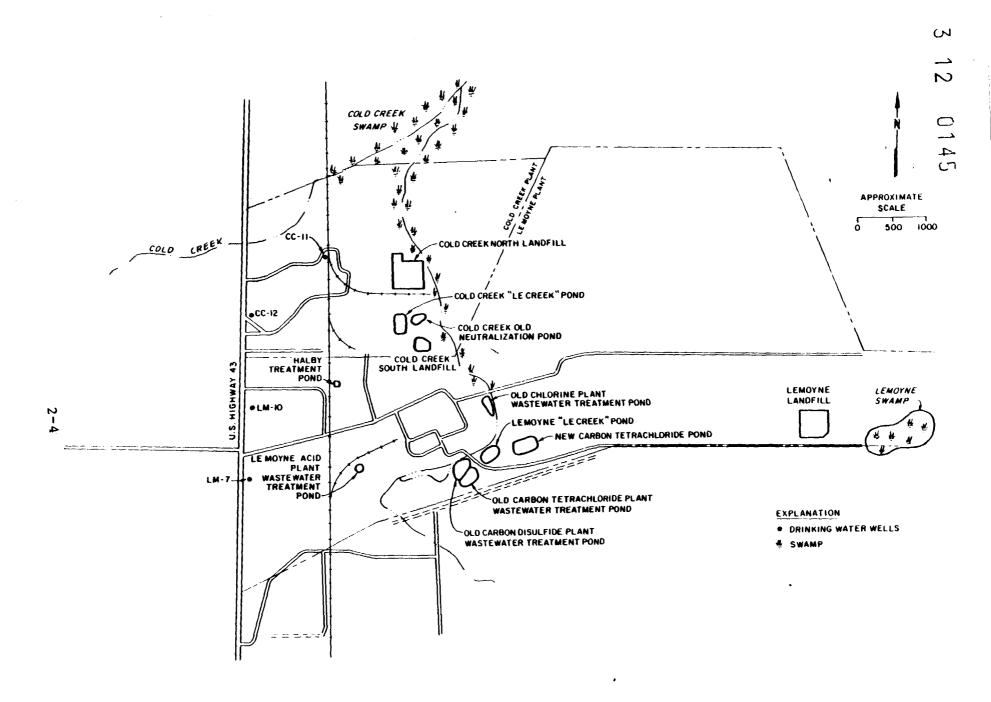


Figure 2-2 Cold Creek/LeMoyne Site Map

membrane-lined and monitored regularly. Of the five inactive wastewater treatment ponds, four (old carbon disulfide plant WWT pond, old chlorine plant WWT pond, Halby treatment pond, and Cold Creek old neutralization pond) are closed and covered. The old carbon tetrachloride plant WWT pond was lined and contains approximately 1900 cubic yards of sulfur sludge; it is inactive but not closed.

Cold Creek swamp received effluent from the LeMoyne and Cold Creek plants as well as from a previous tenant, the Halby Chemical Company (HCC). The effluent from the LeMoyne plant consisted of process waters from production units, containing 10 parts per million (ppm) of mercury. Neutralized waste brine from the Cold Creek plant was also discharged to the swamp during the late 1960's and early 1970's. The contribution from HCC was assumed to have been thiocyanate - contaminated wastewater (Stauffer, 1987).

The LeMoyne swamp may have received surface-water runoff from the area of the LeMoyne landfill prior to its closure in 1974 (Stauffer, 1987).

A small parcel of land on the western portion of the Cold Creek/LeMoyne site was leased from 1965 to 1979 to the Halby Chemical Company (HCC), as noted above. Witco. Inc. purchased the Halby Chemical Company in 1974. Although little is known of this operation. Stauffer (1987) reports that waste products and effluents were discharged to the Cold Creek swamp to the east and/or held in a pond on the property. The HCC buildings were razed in 1979 after the company was purchased by Witco. Inc., and the pond has been closed and filled.

#### 2.2.2 Environmental Contamination and Remediation

During the late 1960's, Stauffer monitored their on-site water supply wells and CNA monitored their production wells for chloride content. By 1971, ground-water monitoring results indicated that chloride concentrations in the wells were increasing, and the ground-water quality was deteriorating.

In October, 1972, an inventory shortage of approximately 10,000 gallons of carbon tetrachloride (CTC) was discovered at the LeMoyne facility storage area. It was thought that some of the CTC seeped into the ground and some flowed into an unlined effluent pond. None of the CTC could be recovered.

By early 1973, Stauffer determined that contaminants were entering the ground water beneath the plant sites, and an in-house task force was organized to evaluate the problem and develop recommendations for remedial action. Twenty-one ground-water monitoring wells were installed, and a swamp-sediment sampling program was initiated. The major contaminants found in the ground water were thiocarbamates, chlorides, and sulfur compounds. Mercury, most likely in the insoluble mercury sulfide form (see Appendix XXV of the Remedial Investigation Report), was found in the swamp sediment samples but not in the ground water. The major sources of these contaminants were determined by Stauffer to be Cold Creek swamp, unlined wastewater holding and treatment ponds, unlined waste-disposal sites containing drummed and uncontained waste materials, process leaks and spills, and leachate from contaminated soils. The predominant areas of ground-water contamination were to the south and east of the site.

By late 1973, Stauffer had initiated contaminant source cleanup activities. Drummed waste debris and sludges in the disposal areas were consolidated and transported off site to permitted hazardous-waste landfills. Liquids were decanted and removed for treatment or off-site commercial disposal. The disposal areas are permanently closed and capped with an impermeable synthetic liner, clay, and soil. Ponds were dewatered, filled, and replaced with lined ponds. Pursuant to an NPDES permit, effluent flow from both facilities was treated and discharged to the Mobile River instead of the swamp. These source control and cleanup activities were completed in 1976.

In addition to the remedial activities described above, an extensive ground-water monitoring program was initiated in December of 1973. Ground-water monitoring continued, and by

late 1977, Stauffer concluded that ground-water quality along the southern property boundary of the LeMoyne facility was improving but at an unacceptable rate. In addition, CNA reported continued contamination of two of their wells, chloride and iron being the primary contaminants. In response to continuing ground-water quality deterioration, Stauffer installed seven new observation wells along the southern property line of the LeMoyne facility.

In December, 1977, leak detection systems for two treatment and storage ponds on site indicated leakage through pond liners. These two treatment and storage ponds were subsequently repaired. In 1978, Stauffer dewatered and capped an old mercury sulfide WWT pond and a WWT pond in the carbon disulfide plant area.

In 1978, Stauffer contracted with Ground Water Associates,. Inc. (GWA) to perform a hydrogeological investigation of ground-water contamination at the LeMoyne facility. During September of that year, GWA collected water samples from 32 wells and analyzed the samples for carbon tetrachloride and carbon disulfide contamination. Based on the analytical results and the results of pump tests, it was concluded that three intercept wells, each pumping 500 gallons per minute (gpm), would be required to intercept the bulk of the contaminated ground water. The three interceptor wells were installed in late 1980 along with an air stripping treatment system approved by the Alabama Water Improvement Commission (AWIC), which is now the Alabama Department of Environmental Management (ADEM). Since that time, the levels of ground-water contamination have been substantially reduced, and treated effluent concentrations have continuously met discharge permit limits established by the ADEM.

At the request of the Alabama Department of Public Health (ADPH). Stauffer in 1980 installed seven new monitoring wells around the two closed Cold Creek landfills. Early in 1981, the leaking treatment and storage ponds at the LeMoyne facility were repaired.

In 1982, the ADPH, in conjunction with the U.S.

Environmental Protection Agency (EPA), conducted an on-site assessment of the Cold Creek and LeMoyne facilities. Analyses of ground-water samples collected during the assessment identified ground-water contamination on the plant sites and at the CNA well that was sampled. The primary contaminants detected were metals, chlorides, carbon tetrachloride and miscellaneous organic compounds. As a result, the Stauffer sites were added to the National Priorities List (NPL) by the EPA under the mandate of the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA), also known as Superfund. The Cold Creek site was ranked number 204 and the LeMoyne site number 467 on the NPL.

Camp Dresser and McKee, Inc. (CDM), under contract to the EPA, conducted a preliminary sampling of the site in May of 1985 and prepared a Work Plan for performance of a Remedial Investigation/Feasibility Study (RI/FS) at the Cold Creek/LeMoyne site. Stauffer performed the RI in 1986 and published a draft RI Report in February, 1987. The objectives of the RI were to define all potential contamination sources and to identify the extent of any contamination migration as well as potential transport pathways. Specific areas investigated were the north and south Cold Creek landfills, the LeMoyne landfill, ponds and lagoons, the Cold Creek swamp, and the ground-water contamination south and east of the site. The results of the RI are discussed in Section 2.4 and are used as the basis for this endangerment assessment.

#### 2.3 Site Hydrogeology

#### 2.3.1 Physiography

The Cold Creek/LeMoyne site is located in the Piney Meadows physiographic province which borders the Mobile River from central Mobile County down to and along the Gulf Coast of Alabama. In the site area, the province is twelve miles wide

and is developed on late Pleistocene river terraces and present day floodplain deposits. These deposits are superimposed on and entrenched into underlying Miocene deposits consisting of sands and clays, with the latter predominating toward the south in Mobile County.

#### 2.3.2 Soils

The soils encountered at the site are loamy clays with loamy clayey subsoils. Locally, poorly drained areas of organic material and mucky clays occur. The major soil type identified by the Soil Conservation Service is the Isagora-Annemaine Association with gentle to moderate undulations (Stauffer, 1987).

#### 2.3.3 Geology

The site is underlain by low river terrace and alluvial deposits that are approximately 110 to 130 feet thick. These deposits thin to approximately 60 feet adjacent to the Mobile River, which is located approximately one and one-half miles east of the central plant area. The deposits consist of generally clean, unconsolidated, fine to very coarse-grained sands that contain some interbedded, discontinuous clayey seams as well as some gravelly zones. The upper sands, varying in thickness from 0 to 50 feet, consist of fine to medium-grained sands, fine-grained sandy silts, silty clays, and clays. The upper sands have moderate to low permeability. The lowermost sands, situated generally between 80 feet below ground surface and the base of the aquifer, contain the most highly permeable material.

A very stiff, dense, bluish-gray clay, presumably of marine origin, underlies the alluvial deposits. The top of the clay unit is from 115 to 130 feet below ground surface in the plant area. The clay unit is more than 20 feet thick, and dips very slightly to the southwest (Stilson, 1974). Previous

studies suggest that the clay unit is continuous across the site and serves to isolate the alluvial deposits from other stratigraphic units below.

GWA (1978) identified an intermediate clay unit in the western sector of the property. They suggest that it becomes discontinuous, thinning to the east into intermittent lenses.

#### 2.3.4 Ground Water

The river terrace and alluvial deposits mentioned above contain a substantial amount of near-surface ground water and are part of the Mobile River Valley water-table aquifer. At the site, this aquifer is recharged through infiltration from the Cold Creek swamp, the Mobile River, and rainfall. The background water quality is good with less than 50 parts per million (ppm) of total dissolved solids, indicating that the water is potable.

Prior to industrialization, ground-water flow was oriented eastward toward the Mobile River. The ground-water table varied in depth from 0 to 20 feet below ground surface depending on the topography. Presently, however, the direction of flow is toward the south-southeast because of the local influence of pumping at the CNA well field and the Stauffer interceptor wells. In addition to effectively causing ground water to flow away from the Mobile River, ground-water pumping has resulted in a lowering of the water table.

#### 2.3.5 Surface Water

Natural drainage from several hundred acres of land, including the western part of the LeMoyne plant property, a portion of the north central part of the adjacent CNA property, and a part of the adjacent Route 43 right-of-way, forms an unnamed stream that flows in an easterly direction south of the LeMoyne plant area. This stream then turns northward and flows generally north-northwest through a 20-acre area of the Cold

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Creek swamp. Flow from the marsh area joins Cold Creek, which flows northeasterly and then easterly to discharge into the Mobile River more than a mile upstream from the eastern extension of the LeMoyne plant property.

Generally the potential for flooding in the site area is minimal. Although high-intensity rain storms (greater than two inches per hour) are not uncommon, they generally do not last for significant lengths of time. The 100-year flood zone in the site area is located in the eastern-most section of the LeMoyne plant property adjacent to the Mobile River. The approximate area covered by the 100 year flood plain is 55 acres. The zone of influence of the 500-year flood plain is only slightly larger than that of the 100-year flood plain. The closed LeMoyne landfill is approximately 500 feet west of and not within the flood plain (Stauffer, 1987).

#### 2.4 Extent of Contamination

The extent of contamination at the Cold Creek/LeMoyne site was determined as the result of the RI. The RI was divided into two major subtasks, source characterization and area characterization. Source characterization included the installation of 3-foot-deep soil borings at 34 locations in the Cold Creek swamp and at 4 locations in the LeMoyne swamp. Of these 38 composite samples, 7 were analyzed for thiocarbamates, chlorides, and priority pollutants, and 31 were analyzed for mercury only.

Soil sampling was also performed under each of the nine ponds and three landfills on site. Soil borings were installed at 45 degree angles beneath the ponds and landfills and advanced to 50 feet or until ground water was encountered. Composite samples were collected at 10-foot depth increments and analyzed for location-specific compounds and priority pollutants. A total of twelve soil borings were installed under the three landfills, and a total of eighteen soil borings were installed under the nine ponds. Water samples were

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collected from three active ponds and analyzed for priority pollutants. In addition, fish samples from five locations in the Cold Creek swamp were collected and analyzed for mercury. Source characterization was completed with the installation and sampling of two new ground-water monitoring wells and sampling of thirteen existing wells. All fifteen well samples were analyzed for priority pollutants; three of the fifteen samples were also analyzed for site-specific compounds.

Area characterization involved sampling 36 site-area wells for location-specific compounds. Seven of the 36 samples were analyzed for priority pollutants. In addition, two surface-water samples and two soil samples were collected offsite to determine background concentrations of the compounds of concern.

Results of the soil, biota, surface-water, and ground-water sampling and analysis conducted during the RI are summarized in Tables 2-1 through 2-3. Only those compounds detected are included in these tables.

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TABLE 2-1. CONTAMINANTS DETECTED IN SEDIMENTS, SURFACE WATER AND BIOTA IN COLD CREEK AND LEWOYNE SHOUPS, MOBILE COUNTY ALABAMA

	_			THEOCARRAMAT	E\$			Makhulasa										$\bigcirc$
Samples	Hereury	<b>EPIC</b>	Butylate	Vernolate	Pebulate	Molinate	Cycloate	Methylene Chloride	Arsenic	Seryilium	Chronium	Copper	Leas	Mickel	line	Yanadzust	Chloride	
Bod   north Samp LS-49 LS-18 LS-25 LS-25 CCS-16 CCS-25 CCS-25 CCS-25 CCS-16 SW-11-16	0. 2800 0. 1100 0. 1500 0. 2200 220, 0000 190, 0000 230, 0000 0. 9000	nd nd nd nd 0.0500 0.2000 1.0000	1, 5000	nd nd nd nd nd 0, 2000 1, 1000	nd nd nd nd nd nd 0, 3000	nd nd nd nd 0, 0500 0, 4000 0, 9000	nd 0, 2000 nd nd nd 0, 5000 1, 8000	0, 0259 nd ne ne nd nd ne	nd nd 8,0000 7,0000 5,0000 5,0000	8.6500 0.4000 0.1100 0.7100 0.4800 0.3100 0.8100	50,0000 19,0000 6,2000 24,0000 120,0000 140,0000 180,0000	8. 3000 6. 4000 1. 5000 10. 0000 13. 0000 14. 0000 34. 0000 68. 0000	12.0000 15.0000 nd 23.0000 35.0000 nd 26.0000	3. 5000 2. 1000 ns 5. 8000 49. 5000 32. 0000 56. 0000	\$5,0000 12,0000 20,0000 131,0000 171,0000 312,0000 561,0000 180,0000		50,0000 190,0000 nd 100,0000 nd 50,0000	0.4
23-2-18 23-2-18 23-1-18 23-3-18 23-3-18 23-4-28 23-4-8 23-4 23-4 23-4 23-4 23-4 23-4 23-4 23-4	1, 8000 59, 0000 29, 0000 38, 0000 1, 2000 2, 0000 md 15, 0000 15, 0000 12, 7000 9, 3000 5, 3000 1, 8000 6, 4000							`										
CS-7-26 CS-7-34 CS-7-18 CS-7-26 CS-7-26 CS-7-26 CS-7-26 CS-7-26 CS-7-26 CS-7-26 CS-8-18 CS-8-16 CS-8-1	5, 6000 6, 9000 7, 7000 101,0000 15, 0000 49, 0000 25, 0000 10, 5000 17, 0000 2, 1000 8, 3000 2, 2000 1, 7000 7, 0000																	
Maximum Averages Frequencys	690, 0000 45, 7308 38/39	1.0000 0.1786 3/7	1, 8000 0, 3000 2/7	1.1000 <b>0.1857</b> 2/7	0.3000 0.04 <b>29</b> 1/7	0.9000 0.1929 3/7	1. 8000 0. 3571 3/7	0, 025 <del>9</del> 0, 0029 1/8	7. 0000 3. 0000 5/8		180, 0000 78, 4000 8/8	68, 0000 21, 9500 8/8	100,0000 26,3750 6/8	56.0000 19.3125 7/8	561, 0000 180, 2500 8/8	-/0	190, 0000 62, 6571 5/7	
Heter Basple Si-01-11 Si-11-11	0.0002							Né	nd	mi	M	ad	mi	rei.	0. 3100	0.0002		
Maximum; Average; Frequency;	0.0002 0.0002 1/1	-/0	-/0	-/0	-/0	-/0	-/0	0,0000 0,0000 0/1	0,0000 0,0000 0/l		0,0000 0,0000 0/1	0,0000 0,0000 0/L	0,0000 0,0000 0/1	0.0000 0.0000 0/1	0.3100 0.3100 1/1	0.0002 1/L	-/0	
Fish Samples 19-1 19-2 19-3 19-4 19-5	0. £300 3. 1000 0. 9100 1. 1000 0. 4200																	
Renimum Averages Frequencys	1.1000 1.2320 5/5										•							

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All Concentrations are in pps Average =  $Cl_{-1}$ ,  $Cl_{-1}$ ,  $Cl_{-1}$  and  $Cl_{-1}$  and  $Cl_{-1}$  are in a supplex  $Cl_{-1}$ . A supplex  $Cl_{-1}$  are the supplex share compound was detected/Total number of samples analysed. The average of the two samples were taken for duplicate samples.

r831.0M	Puteringe	Maximum Average: Frequency:	ge;	001		D D D D D D D D D D D D D D D D D D D		000 64 101	000 \$5.7	日 日 日 日 日 日 日 日 日 日 日 日 日 日 日 日 日 日 日	600.4-7		20-30 -73	33-13 3-15-23	20-44-E	22-2- 22-3- 25-21-21-21-21-21-21-21-21-21-21-21-21-21-		3899 747	22		- u	799 820		800 100 100 100 100 100 100 100 100 100	900-017-12 900-017-12	<b>F</b>	
All concentrations Average • Cl • C2 Fraquency • Number The average of the	meter wells	10.420 10.420 14/11	0.00%	tan 1	0.0070	2.01%	Z	9. 9097	0.0145	2			Z	Z		i	1 oi	. I	0.0012 0.0012	e <b>a</b>	0.0020 0.0020	e ivi				Tetrachloride	Carbon
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pies where of		0.1120 0.01020 0.01200	2 P	R												i	L L	t.					LL	ZZi	LZZ	Cytalds	
are in pen /lotal no. of samples  of samples where compound was detected/Total number of samples analyzed.  two samples was taken for duplicate samples.		0.000 <b>8</b> 0.000 <b>1</b> 2/13	Z .000	0.0001												i	LLI	Ł					ZZ	EEI	LEE	- Lease	
letected/Total		1.2000 0.0270 20/51		0,0070	EZ	PZ Z	222	ZZ	eri R	LZZi	125	99 39	ZZ	ZZ	<b>PZZ</b>	1.2000	351	9.00	121	0.0021 0.0021 0.0021	0.0036		. M	PE 1	l a	eg at	
il number of		0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.0		0.0021	22	21 E	ZZZ	11	222	LZZi	i i	212	ZZ	22;	121	22;	2 2 5	0.00 0.00 0.00 0.00	2 9 1 2 8 2 1	1 2 8	0.002J	200 200 200 200 200 200 200 200 200 200	0.00L	ZZi		Butylate 1	9
samples and		0.0087 0.0010 14/31		0.0020	ZZ	P Z Z	ZZZ	22	ZZZ	ZZZ		e i	. 2 2	22;	KKE	222	<b>3</b> 5.	0.0 0.0 0.0 0.0 0.0 0.0 0.0 0.0 0.0 0.0	<u> </u>	0.0010 0.0010	9.00 20.00 2	200	ZZ	R Z Z	Z	Vernolate	THI OCCHABONATES
y2 <b>m</b> 2.		0.0019 0.0001		Zi	LZi	122	ZZZ	ZZi	I I I	ZZZ		ZZ	ZZ	ZZi	LZZ	111	L	. <b>Z</b> Z i		2.9 0 0	ZZi	<b>5</b> 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5	ZZi	r r z	.Z	Pebulate	
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		0.020 0.0030 11/2	118	l												3	1.02 0.02 0.02 0.02					i	i gi			Branic	
		0.0036 0.0036	221	L												2	* 2					i	r z e	628 628	22	Copper	
		0.0130 0.0093 7/13	0.0230	l												Z	0. 01.20 0. 0336					i	2.0.5 2.0.5 2.0.5	22	3.5	Mickel	
		0. 1700 0.0725 13713	90 90 90 90 90 90 90 90 90 90 90 90 90 9	• }												6.059	9 9 03 03 00 00 00							0.1700 0.2700	0.06 04 04 04 04 04	Ä	
		3900,0000 141,7666 51/51		\$1.7000	£ 2000	29. 7000 4000	11. 5600 2000 2000 2000 2000	17.8000	14. 1000 14. 1000 14.	J7. 9000 J7. 9000	\$6. 7000	17. 4000	F 9000		37.8000	22. 1000 1000	91.3000 0006.52	159. 3000 233. 0000	51. 2000 5000	367.0000	000£.04 000£.04 000£.04	J90, 0000	5.3000 5.3000	9. 7000 5. 0000		Chloride B	
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TABLE 2-2. CONTANTMENTS DETECTED IN GROUND WATER AT THE COLD CREEK/LDIOYNE STIE, MOBILE COUNTY ALABAMA

9910 71 2 9910 71 2



TABLE 2-3. CONTAMINANTS DETECTED IN COMPOSITE SOIL SAMPLES IN PONDS, LAGOONS, AND LANDFILLS AT THE COLD CREEK/LEMOYNE SITE, MODILE COUNTY, ALARAMA

Sample: Ponds:	Cyanide	Mercury	Hethylene Chloride	Tolugno	Fluoranthene	Pyrune	Antimony	Arsenic	Beryllius	Cadma van	Chronium	Copper	Lead	Nickel	Selenaue	Thailsum	linc	Variad sud	7
Men CTC Po MCTC-19 MCTC-29	nd nd nd	nd ne	1.0600 2.9900	2	nd nd	nd Nd	nd nd	4.0000 nd	0. 1300 0. 1500	nd nd	29. 0000 100. 0000	4. 0000 4. 6000	5. 8000 Nd	1. é000 8. 2000	nd nd	nd nd	15.0000 21.0000	1 ;	S
MisHoyne Po LLP-19 LLP-25	nd nd nd	nd nd	1 1400 1 1600	M	<b>1</b>	M M	nd nd	nd nd	0.0610 0.2100	nd nd	6. 5000 10. 7000	2. 2000 1. 2000	5. 4000 6. 5000	1.7000 2.5500	M M	nd nd	10.0000 11.5000		0
COID Creek CCAP-18 CCAP-28	Pond 0,5000 nd	nd rel	M M	M M	nd nd	ni N	nd nd	2.0000 1.0000	0.0865 0.1200	nd nd	8. 4500 7. 4000	2. 9000 2. 3000	nd nd	1.4000 1.6000	nd nd	nd nd	7. 9560 5. 3000		Ω.
Acid Plant APP-19 APP-29	MAT Pond nd nd	nd nd	M M	*	nd mi	rd rd	nd nd	1.0000 1.5000	0. 2000 0. 1600	nd ná	12.0000 10.4500	4. 2000 3. 7500	7. 2000 7. 7 <b>5</b> 00	1.2000 1.2500	nd Nd	0, 5000 nd	14.0000 8.7500		
Did CoPlant MTP-18 MTP-28	hill Pond nd nd	1.4000 24.0000	*	<b>N</b>	nd nd	nd nd	nd Nd	rd rd	0.0430 Mi	nd NG	4.5000 8.6000	2. 3000 1. 3000	4. 9000 5. 8000	nd 1.6000	<b>M</b>	nd nd	9.7000 12.0000		
014 CTC W/T 02TC-18 0CTC-29	Pond nd nd	nd 0. 3000	0.9700 nd		0. 1625 M	0. 1345 M	7. <b>65</b> 00 31. 0000	nd 1.0000	nd 0. 0780	nd nd	3.2500 15.0000	12. 2000 1. 4000	nd nd	1.2500	nd ad	0. 2500 nd	5.5000 11.0000		
01d C92/CTC 0C9-18 0C9-26	MAT Pond nd nd	0. 4000 nd	14 14	M M	nd nd	M	32.0000 26.5000	7. 0000 2. 5000	0.0980 0.1200	nd nd	14.0000 14.5000	15, 0000 9, 6500	nd nd	2.6000 8.7000	nd nd	nd nd	15.0000 24.5000		
LaCreek MAT CCLP-29 CCLP-19	Pond nd nd nd	nd rd	1.2850 76	nd nd	nd në	nd nd	nd nd	2.0000 1.0000	0. 3000 0. 4500	nd nd	12.6000 15.0000	4. 1500 5. 0000	nd nd	2. 8000 1.6000	nd nd	nd ns	12.5000 21.0000		
Melby HAT P HTP-25 HTP-15 HTP-38	rond Rd 240,0000 NG	nd 0. 9000 nd	M M M	12 12 13	nd nd nd	76 76 76	nd nd nd	0.5000 2.0000 1.0000	0.1100	nd 1. 6000 Nd	13.5000 41.0000 15.0000	4.8500 442.0000 4.5500	2. 2000 22. 0000 nd	3. 2000 18. 0000 3. 4500	nd nd nd	nd nd nd	32,0000 1170,0000 25,0000		
Landfills																			
Lefloyne Ler LLF-15 LLF-25 LLF-35 LLF-46 LLF-58 LLF-60 LLF-75 LLF-80	edfill ed	0. 1000 nd 0. 1000 nd nd 1. 9000 0. 1000 nd	nd nd 0, 9700 nd 0 0, 7430	100 100 100 100 100 100 100 100 100 100	nd nd nd nd nd nd nd	nd nd nd nd nd nd nd	25. 0000 7. 5000 23. 0000 33. 0000 16. 0000 11. 0000 nd 8. 6000	nd nd nd nd 1,0000 nd nd nd	0. 0290 nd 0. 2400 0. 0550 nd nd nd	100 100 100 100 100 100 100 100 100 100	48. 3000 5. 3000 15. 0000 22. 0000 7. 8000 7. 1000 3. 8000 5. 2000	54. 5000 7. 2000 23. 0000 35. 0000 26. 0000 19. 0000 15. 0000 32. 0000	S. 2500 rel rel rel rel rel rel rel	3, 6500 1, 3000 3, 6000 3, 9000 1, 6000 4, 1000 nd 1, 9000	nd 0.5000 nd nd nd nd 0.5000 nd	nd nd nd nd	13, 6000 6, 8000 19, 0006 22, 0000 7, 1000 14, 0000 4, 4000 8, 1000		
North Drum NLF-28 NLF-18	limid Landfil nd nd	0. 6000 nd	nd nd	nd nd	nd nd	nd nd	nd Ne	1.0000 nd	0. 1450 0. 1700	nd Nd	14,0000 6,5000	2. 9500 2. 5000	nd na	1.5500 1.4000	no nd	nd nd	10. 3000 <b>å.</b> 9000	21.0000 8.3000	
Bouth Drum SLF-15 BLF-28	field Landfil nd nd	i nd nd	Nd Ad	rei nei	nd nd	ná ná	ná ne	1.0000 2.5000	9. 2100 9. 7050	né Mi	16, 0000 34, 5000	4.5000 6.2000	4.0000 nd	2.1000 5.3000	0.5000 0.6200		15, 0000 35, 2000	26.0000 51.5000	
Maximumi Averages Frequencys	240, 0000 7, 7581 2/31	24,0000 0,9313 10/31		3. 46/ 0. 17/ 1/31	00 0, 1625 61 0, 0052 1/31	0. 1345 0. 0043 1/31	33. 0000 7. 1694 11/31	7,0000 1,0323 17/31		1.6000 6.0516 1/31	100, 0000 17, 1339 31/31	442.0000 24.6323 31/31	22,0000 2,4774 11/31	18,0000 3,2419 29/31			1170,0000 44,3606 31/31	51,5000 26,7000 4/4	

HICTIVE MAGTEMATER TREATMENT PONDS

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All concentrations in ppm. Average  $\sigma$  C1 + C2 +.../folal number of samples. Frequency = Number of samples where compound was detected/Total number of samples analyzed. The average of the two samples was taken for duplicate samples.

#### 3. ENVIRONMENTAL FATE OF INDICATOR COMPOUNDS

The results of the RI were used by Stauffer (1987) to preliminarily assess potential public health and environmental impacts posed by the site. Based on the concentrations detected, the frequency of detection, background levels, and the toxicology of the compounds detected, eleven contaminants were considered in the preliminary endangerment assessment to determine the potential risks to humans and wildlife in the area. These eleven compounds, referred to as indicator compounds, include carbon disulfide, carbon tetrachloride, cyanide, mercury, thiocyanate and six thiocarbamates, cycloate, EPTC, butylate, molinate, pebulate, and vernolate.

Table 3-1 shows the maximum and average concentration of each indicator compound detected in the different media. The table also shows the frequency at which the compound was detected (i.e., the number of samples in which the compound was detected compared to the number of samples that were analyzed). The environmental fate and transport of each of the indicator compounds is described below.

#### 3.1 Carbon Tetrachloride

Volatilization is the major transport process for carbon tetrachloride in aquatic systems because of its high vapor pressure. There is little information specifically pertaining to adsorption of carbon tetrachloride onto sediments.

McConnell et al. (1975) found that coarse gravel had little adsorptive capacity for carbon tetrachloride, whereas sediments rich in organic detritus had a much higher adsorptive capacity. In general, however, there is no clear evidence of selective concentration of carbon tetrachloride in sediments.

Neely et al. (1974) have shown that bioaccumulation is directly related to the logarithm of the octanol/water partition coefficient (log  $K_{OW}$ ) of the compound. The log  $K_{OW}$  of carbon tetrachloride is 2.64, indicating a tendency



TABLE 3-1. SUMMARY SHEET FOR INDICATOR COMPOUNDS AT THE COLD CREEK/LEMOYNE SITE, MOBILE COUNTY, ALABAMA
(all concentrations are in ppm)

	•	Swamp Sediments	ents		Swamp Water			Swamp Biota	•
	Maximum	Average	Frequency Maximum	Maximus	Average	frequency Maximum	Meximus	Average	Frequency
Carbon Tetrachloride			0/9	•	•	-/0	•	•	0/1
Carbon Disulfide	•		-/0	•	•	-/0	•		./0
cyanide	,	•	0/8	•		./0		•	-/0
Mercury	690.0000	45.7308	38/39	0.0002	0.0002	3	3.1000	1.2320	5/5
Thiocarbamates									
ЕРТС	1.0000	0.1786	3/7	•		· •		•	-/0
Butylate	1.8000	0.3000	2/7	•		۰,	•	•	-/0
Vernolate	1.1000	0.1857	2/7	•	•	-/0			-/0
Pebulate	0.3000	0.0429	1/7	•	•	-/0	•	•	. 0/.
Mol inate	0.9000	0.1929	3/7	•	•	· •	•	•	./0
Cyclosts	1.8000	0.3571	3/7	٠	•	%	•	•	-/0
Thiocyanate	•		-/0		•	./0		•	./0
	Groundwater (all wells)	(all wells)		Groundweter	Groundweter (downgredient wells)	or wells)	Ponds, Lagoons, Landfill Soils	ons, Landfi	lt Soils
	Mex i mus	Average	Frequency	Maximum	Average	frequency	Maximum	Average	Frequency
Carbon Tetrachloride 298.0000	298.0000	10.4280	18/33	0.2245	0.0332	6/8	685.0000(a)		
Carbon Disulfide	55.6000	1.5385	20/51	0.0454	0.0030	9/19	15.0000(a)		
Cyanide	0.1320	0.0102	1/12	•	•	0/2	240.0000(b)	7.7581	2/31
Mercury	0.0008	0.0001	2/13	•	•	0/2	24.0000(b)	0.9313	10/31
Thiocarbamates									
€PTC	1.2000	0.0270	20/51	0.0210	0.0016	3/19	0.9000(*)		
Butylate	0.0135	0.0010	14/51	0.0068	0.0005	2/19	0.2000(c)		
Vernolate	0.0087	0.0010	14/51	0. 1700.	0.0005	2/19	0.1000(c)		
Pebulate	0.0019	0.0001	2/51	٠	•	0/19	•		
Hol inste	0.2310	0.0081	20/51	0.0260	0.0029	3/19	1.5000(a)		
Cycloste	0.0071	0.0008	13/51	0.0057	0.0004	2/19	0.9000(a)		
Thiocyanate	4.0000	1.0667	2/4			•	2560.0000(a)	•	
Notes:									

Notes:

(a): Sample taken 10 feet below surface
(b): Composite sample
(c): Sample taken 20 feet below surface

for this compound to bioaccumulate under conditions of constant exposure. Pearson and McConnell (1975), found that although carbon tetrachloride and other organochlorines examined are somewhat lipophilic and tend to be found at higher concentrations in fatty tissues, there is no evidence for the biomagnification of carbon tetrachloride or other short-chain aliphatics in the food chain. According to the EPA (1979), however, the difficulties associated with the analytical methods in the Pearson and McConnell (1975) study make estimates of bioaccumulation based on their experimental results somewhat unreliable.

There is little information specifically pertaining to biodegradation of carbon tetrachloride. Thom and Agg (1975) have included carbon tetrachloride on a list of synthetic organic chemicals which should be degradable by biological sewage treatment methods provided suitable acclimatization can be achieved. They note, however, that not many compounds on the list occur free in nature, and, therefore, it is unlikely that microorganisms already possess the ability to destroy them.

#### 3.2 Carbon Disulfide

Very little information is available regarding the transport and fate of carbon disulfide. Nevertheless, data regarding the physiochemical properties of carbon disulfide (Verschueren, 1983) can be used to qualitatively assess the processes that may be important in determining its fate. Carbon disulfide has a relatively low boiling point and high vapor pressure, indicating that it is very volatile. It has a specific gravity of 1.263 and is therefore more dense than water. With a solubility of 2300 mg/l, it is very soluble in water from an environmental perspective. The octanol-water partition coefficient of carbon disulfide is in the range of from 69 to 145, indicating that it is not readily adsorbed to soils. Given this information, it is likely that

volatilization and ground-water mobility are significant whereas adsorption is of lesser importance in terms of the transport of carbon disulfide. The significance of photolysis, hydrolysis, bioaccumulation, and biotransformation cannot be assessed with the information available from Verschueren (1983) regarding the chemical and physical properties of carbon disulfide.

#### 3.3 Cyanides

Cyanides are a diverse group of compounds whose fate in the aquatic environment varies widely. The cyanide ion (Cn) can react with a variety of metals to form insoluble metal cyanides. If the cyanide ion is present in excess, complex metallocyanides may be formed. These compounds are quite soluble and can be transported in solution. The fate of low molecular weight organic cyanides (nitriles) is expected to parallel the fate of hydrogen cyanide, a gas which may be destroyed by biodegradation or removed from solution by volatilization or adsorption.

The significance of photolysis on the aquatic fate of the cyanides has not been investigated fully, although it is possible that the photolysis of the metallocyanides could result in the release of cyanide ion (Broderius, 1977). EPA (1979) notes that this process could be important in aquatic environments downstream form metallocyanide discharges.

According to the EPA (1979), the hydrolysis of nitriles in the aquatic environment is slow in most cases, and is probably not competitive with other processes.

Cyanides are fairly mobile in the soil environment (Alesii and Fuller, 1976), indicating that adsorption is probably not a significant control on mobility in most aquatic environments where sorbents (e.g., clays, biological solids, sediments) are much less concentrated. Alesii and Fuller (1976) reported that cyanide mobility is least where soils have a low pH, high concentrations of free iron oxides and positively-charged

particles such as the clay minerals Kaolin, chlorite, and gibbsite. Cyanide mobility is greatest at high pH, high concentrations of free calcium carbonate (high negative charge), and low clay content. According to the EPA, biological solids sorb cyanides, but, as with the other sorbents, the amount bound is probably insignificant in comparison to the amounts volatilized or biodegraded.

Broderius (1973) reported the bioaccumulation of metal (copper and silver) cyanide complexes in fish. According to the EPA (1979), it is difficult to assess the environmental importance of metal cyanide bioaccumulation other than to note that the metal cyanides are generally less toxic than hydrogen cyanide. Nevertheless, bioaccumulation undoubtedly enhances the chronic toxic effects of the metal cyanides.

Although biodegradation of cyanides is known to occur in natural waters, the rates of cyanide biodegradation have not yet been ascertained. According to the EPA (1979), the importance of this process varies according to such factors as cyanide concentrations, pH, temperature, microbe concentration and acclimatization to cyanide, and the availability of nutrients.

#### 3.4 Mercury

The major removal mechanism of mercury from natural water systems is adsorption onto the surfaces of particulate phases and subsequent settling to the bed sediment. According to the EPA (1979), the overwhelming majority of any dissolved mercury is removed in this manner within a relatively short time, usually in the immediate vicinity of the source.

Photolysis seems to be significant in the chemical speciation of mercury in the atmosphere and perhaps in the aquatic environment. Because of the limited amount of information available regarding this process, however, it is not clear what impact it may have on the overall fate of mercury in the aquatic environment. Because of its uniquely

high vapor pressure relative to other metals, metallic mercury can enter the atmosphere from the aquatic environment in the form of several different gaseous compounds. This factor makes volatilization important for the aquatic fate of mercury. It is not clear what impact volatilization will have on the overall fate of mercury in the aquatic environment, however, because of the limited quantitative data available regarding the volatilization of mercury compounds from natural waters.

EPA's (1979) review of environmental studies and theoretical considerations indicates that mercury adsorption onto sediments is probably the most important process in determining the fate of mercury in the aquatic environment. Carr and Wilkness (1972) found that radioactive mercury, when added to natural samples, was rapidly apportioned out of the particulate phases with half-lives for adsorption ranging from . less than one to fifty hours. Ramamoorthy and Rust (1976) conducted laboratory studies on mercury sorption and found that sorption rates were highest in organic-rich sands, that sediment binding capacity was most closely related to organic content, and that mercury sorption was little affected by pH. In their study of mercury adsorption on and desorption from sediments, Reimers and Krenkel (1974) found that, at a constant pH, the adsorption of inorganic mercury is affected by aquatic chloride concentration, with the percent loss in capacity depending upon the constituents of the sediment. They also found that inorganic mercury is bound strongly enough by sediments to be transported by sedimentary mobilization.

Because of the concern regarding the danger to human health from eating mercury-contaminated fish, the bioaccumulation of mercury in the aquatic environment has been well studied. Methyl mercury is the form of mercury present in most fish tissue, and it is the most readily accumulated and retained form of mercury in aquatic biota. Methyl mercury is readily accumulated by fish both from their food and through water, and is very difficult to eliminate after entering the biological system. Most studies report that the half-life of

methyl mercury in aquatic organisms is between one and three years.

As an element, mercury is not intrinsically altered by chemical reaction, but it does take part in biologically-mediated reactions which drastically alter its mobility and toxicity. According to the EPA (1979), virtually any mercurial compound can be microbially converted to methyl mercury upon entering an aqueous system. Conditions reported to enhance the methylation process include large amounts of available mercury, large numbers of bacteria, absence of strong complexing agents such as sulfide, circumneutral pH, high temperature, and a moderately aerobic environment. (As discussed in greater detail in Appendix XXV in the RI. conditions at the Cold Creek/LeMoyne site did not favor the formation of methyl mercury. Rather, conditions favored the precipitation of insoluble mercury sulfide because of the presence of the strong complexing agent, sulfide.) Bacteria can act not only as mediators of methylation, but can also preferentially accumulate large amounts of mercury. Although sediment is probably the most important sink for mercury, methylation by bacteria could reduce the mercury content of overlying waters, resulting in the mobilization of inorganic mercury from the sediments. In summary, mercury can be metabolized by bacteria to methyl and dimethyl forms which are quite mobile in the environment.

#### 3.5 Thiocarbamates

Very little information is available regarding the transport and fate of the individual thiocarbamate compounds, or the thiocarbamate compounds as a class. Nevertheless, data from the Handbook of Environmental Data on Organic Chemicals (1983) regarding the physiochemical properties of the thiocarbamates have been used in this report to qualitatively assess the processes that may be important in determining their fate. In addition, the limited amount of information available

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regarding the known properties of the individual compounds is reported here.

In general, the thiocarbamates have relatively low to moderate boiling points and moderate vapor pressures, indicating that they are somewhat volatile. Their aqueous solubilities range from 30 to 370 ppm, which indicates that they are soluble to very soluble in water. Given this information, it is likely that ground-water mobility is a significant factor in the fate and transport of the thiocarbamates. Because octanol-water partition coefficients were not available for these compounds, the importance of adsorption and bioaccumulation could not be assessed. In addition, the significance of photolysis, hydrolysis, and biotransformation cannot be assessed with the information available from Verschueren (1983).

Limited information regarding the fate of S-ethyl dipropylthiocarbomate (EPTC) and molinate was available in the Handbook of Environmental Data on Organic Chemicals (1983). Verschueren reports a 75% to 100% reduction of EPTC from soils in four weeks time as the result of "aquatic reactions". According to Verschueren, molinate added to tapwater decreased over a fourteen-day holding period to 40% based on recovery of isotopic carbon (14C). The loss of 14C was attributed primarily to volatilization. The five major organosoluble metabolites in the tapwater were molinate sulfoxide, 3- and 4-hydroxymolinate, 4-ketomolinate, and ketohexamethylene-imine. In addition, Verschueren reports that two kinds of microorganisms isolated from garden soils and rice-field drains degraded molinate completely into various hydroxy and oxidized products. Thus, it has been shown that volatilization and biodegradation are significant in terms of the fate and transport of at least one thiocarbamate.

3 12 0165 3.6 Thiocyanate

There is virtually no information available regarding the fate and transport of thiocyanates. According to the EPA (1979), thiocyanates (SCN radical) are formed from cyanides and sulfur-containing materials, and are more stable than cyanates (OCN radical). Solutions of thiocyanates form free hydrogen cyanide in acidic media.

#### 4. TOXICITY ASSESSMENT

## 4.1 Toxicity Profiles

This section contains toxicity profiles for the indicator compounds at the Cold Creek/LeMoyne site. The dose-response assessment takes two forms, that for noncarcinogens and that for carcinogens. For noncarcinogenic substances, the underlying presumption is that a threshold for the effect exists. That is, there is a dose below which no effect will occur. Acceptable Intakes for Chronic exposures (AICs) are developed by EPA for noncarcinogenic compounds to provide reasonable certainty that the specified intake value is below the threshold and the risk is, therefore, practically zero.

Carcinogens are assumed to have no threshold for adverse . health effects. It is assumed that any dose, no matter how small, of a carcinogen or suspected carcinogen will pose some excess risk of cancer. Only a limited number of chemical compounds have been demonstrated unequivocally to be human carcinogens. However, experimental and epidemiologic data are available that are suggestive of the carcinogenic activity of certain compounds. The quality and quantity of these data vary between compounds. EPA has developed a "weight-of-evidence" system that is intended to reflect the decreasing level of certainty that a compound is, in fact, a human carcinogen based on available data. The categories are:

- 1) A; human carcinogen demonstrated human carcinogen.
- 2) B-1; probable human carcinogen limited evidence from human studies.
- 3) B-2; probable human carcinogen sufficient evidence from animals studies and inadequate evidence from human studies.
- 4) C; possible human carcinogen limited evidence from animal studies

- 5) D: not classified inadequate evidence from animal studies
- 6) E; no evidence of carcinogenity in humans no evidence in human and/or animal studies.

The carcinogenic potential of approximately 200 compounds have been reviewed by the EPA Carcinogen Assessment Group (CAG). CAG has developed computerized methods that extrapolate observed dose-response relations to the low dose levels encountered in environmental situations. They incorporate both the no-threshold assumption and a further assumption that carcinogenic dose-response is linear at low doses. The result of the dose-response curve fitting computations is a 95% upper bound "potency estimate", which has units of reciprocal milligrams of compounds per kilogram body weight per day ([mg/kg/day])<sup>-1</sup>). Assuming linearity a predicted intake needs only to be multiplied by the 95% upper bound of potency estimate to give (unitless) upper bound risk values. The computed risk value should be viewed as an estimate of the excess chance of getting cancer, above background cancer rates, produced by intake of carcinogenic contaminants. Further, the cancer risk estimate for exposure at a site is an "upper-bound" estimate, the actual risk may, in fact, be lower.

#### 4.1.1 Carbon Tetrachloride

Carbon tetrachloride is a multi-purpose chlorinated aliphatic hydrocarbon. Agricultural applications include its use as a grain fumigant and pesticide. Because of its excellent solvency properties, it was used as spot remover until 1970 when the Food and Drug Administration (FDA) banned it (IARC, 1979). Carbon tetrachloride's use as a fumigant has been restricted to seed and grain fumigation purposes only under the Federal, Insecticide, and Rodenticide Act (FIRA) (Federal Register July 27, 1983:34077). The majority of carbon tetrachloride is employed in the production of

chlorofluorocarbons. Refrigerants, foam-blowing agents, and solvents are the most common uses for chlorofluorocarbons and accounted for 95% of the total consumption of carbon tetrachloride in 1980 (EPA, 1984a).

This haloalkane has a vapor pressure of 91.3 mm Hg @ 20°C, is relatively insoluble in water, and is miscible in alcohol, ether, benzene, and many of the fixed and volatile oils (Hawley, 1981). The logarithm of the octanol/water partition coefficient (log K<sub>ow</sub>) for carbon tetrachloride is 2.64 (Leo et al., 1971). These chemical characteristics indicate that carbon tetrachloride is fat soluble and may bioaccumulate.

Neely et al. (1974) used the partition coefficient to estimate the bioconcentration potential of carbon tetrachloride in Salmo gairdnen Richardson (rainbow trout). The estimated logarithm of the bioconcentration factor (BCF) was 1.24, indicating that carbon tetrachloride may bioconcentrate in fish. EPA (1980) reported that a measured steady-state BCF of 30 was obtained using Lepomis machrochirus (bluegill).

EPA (1980) reports that acute toxicity values for freshwater species have been observed at concentrations as low as 35.2 mg/l, and in saltwater species at concentrations as low as 50 mg/l. Data are not yet available to assess chronic toxicity of carbon tetrachloride to freshwater aquatic life.

EPA has classified carbon tetrachloride as a probable human carcinogen (B-2). When ambient water quality criteria are based solely on the potential for adverse health effects, water concentrations should be zero. Because a zero level may not be technologically feasible, lifetime incremental risks of cancer are calculated by the EPA. For an estimated risk of  $10^{-5}$ ,  $10^{-6}$ , and  $10^{-7}$ , the corresponding recommended criteria are 4.0  $\mu$ g/l, 0.40  $\mu$ g/l, and 0.04  $\mu$ g/l, respectively. These values are based on daily consumption of 2 liters of contaminated water and 6.5 grams of contaminated aquatic organisms.

<sup>\*</sup>The non-threshold assumption is made for carcinogens because it is believed that there is no dose level below which an adverse effect (i.e., cancer) could not occur.

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Absorption of carbon tetrachloride through dermal, inhalation, and ingestion routes has been studied in a variety of animal species and in humans. Because of carbon tetrachloride's lipophilic nature, it is readily absorbed through the skin. Stewart and Dodd (1964) conducted a series of human absorption experiments where the skin of the hands of volunteers was exposed to several chlorinated aliphatic hydrocarbons. Carbon tetrachloride not only penetrated the skin, but it was also likely to be "absorbed through the skin in toxic quantities when in contact with the skin of the hands and forearm" (Steward and Dodd, 1964).

> The EPA (1984a) has reviewed the metabolism of carbon tetrachloride and proposed mechanisms of toxicity. These are briefly outlined below. A large percentage of carbon tetrachloride is absorbed when inhaled, whereas a smaller percentage is absorbed when ingested. Distribution of carbon tetrachloride is body wide with the highest concentrations found in fat tissues, liver, bone marrow, and blood (EPA. 1984a). Carbon tetrachloride is metabolized into chloroform and a trichloromethyl radical in the liver.

The acute toxicity of carbon tetrachloride involves primarily the liver, kidney, and lungs. At lower doses liver enzymes are disturbed in mice and dogs (Klaassen and Plaa, 1966, 1967; Sein and Chu, 1979). In both chronic and subchronic toxicity studies, fatty liver infiltration, inflammation, and cell death has been observed. The extent of these histopathologic conditions depends on the dose administered. Bizin et al. (1977) compared continuous inhalation of carbon tetrachloride (i.e., 500 mg  $CCl_A/m^3$ air for 10 consecutive days) to intermittent exposures (i.e. same concentration for 6 hours/day for 40 days). Continuous exposure produced toxic symptoms four to five times as rapidly as did intermittent exposure. Pathology of the kidney in mice and rats also occurs but requires higher doses (Plan and Larson, 1965). A dose of 3260 mg/kg body weight administered to mice induced renal dysfunction within 96 hours.

Cellular damage also occurs in lungs at ingested doses of 4000 mg/kg body weight in mice (Gould and Smuckler, 1971; Boyd et al., 1980). Oral doses of less than 1600 mg/kg did not appear to induce pulmonary lesions, whereas dose-dependent cellular damage occurred in the range of 2400 to 4800 mg/kg (Boyd et al., 1980).

Chronic inhalation studies have been conducted in rats, Guinea pigs and rhesus monkeys (Smyth et al., 1936). As was noted in the EPA (1984) review of chronic data, Guinea pigs were observed to be more sensitive to the toxic effects of carbon tetrachloride. Rats suffered growth retardation at the highest dose (2520 mg/m³ - 8 hours/day, 5 days/week for 10.5 months). Cirrhosis of the liver was observed in the three highest doses (630, 1260, and 2520 mg/m³). Additional hepatic changes developed and did not resolve until 156 days post-exposure. Guinea pigs also developed cirrhosis and hepatic surface alternations at the lowest dose (315 mg/m³) administered [to both groups of animals]. Sciatic nerve damage occurred in half (2/4) of the exposed monkeys exposed to 1280 mg/m³.

Although Schwetz et al. (1974) reported some statistically significant retarded growth development in rat fetuses, the authors could not make conclusions regarding the teratogenic potential of carbon tetrachloride because the doses administered (1890 and 6300 mg/m³) were toxic to the maternal rats. The EPA (1984) reviewed several other studies and concluded the methodologies employed in the studies did not "meet current design criteria for hazard assessment purposes."

Several mutagenicity studies have been conducted including seven bacterial assays, one yeast assay, one in vitro mammalian chromosonal damage, and three tests examining in vivo DNA damage in rodents (EPA, 1984). The EPA (1984) noted that although the majority of these assays were negative there was evidence supporting the classification of carbon tetrachloride as a weak mutagen. The production of free radical product and its ability to covalently bind to cellular macromolecules was hypothesized as a mechanism of mutagenicity (EPA, 1984).

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Carbon tetrachloride is classified as a group "B2" carcinogen in the EPA's weight-of-evidence ranking scheme (SPHEM, 1986). This category indicates that there is sufficient animal evidence supporting the carcinogenicity of carbon tetrachloride but that the corresponding human data is inadequate. In developing this ranking, EPA (1984a, 1984b) reviewed oral bioassays for carcinogenicity in various rodent species. The National Cancer Institute (NCI) study (1976) used carbon tetrachloride as a positive control in its investigation of the carcinogenicity of trichloroethylene in rats. Hepatocellular carcinomas and neoplastic nodules occurred in both sexes. Della Porta et al. (1961) observed liver-cell carcinomas in half of each sex (5/10) of the exposed hamsters that survived ten weeks or more after the cessation of treatment. Hepatocellular carcinomas have been observed in additional rat studies conducted by Reuber and Glover (1967, 1970) and in mouse studies conducted by Edwards (1941), Edwards et al. (1942). Because of carbon tetrachloride's carcinogen classification, the EPA (SPHEM, 1986) has not calculated an acceptable intake for either subchronic (AIS) or chronic (AIC) exposure. The oral carcinogenic potency factor has been calculated, however, and is 1.30 x  $10^{-1}$  (mg/kg/day)<sup>-1</sup>.

#### 4.1.2 Carbon Disulfide

Carbon disulfide has many chemical and industrial applications. Its primary use is in the manufacture of regenerated rayon and cellophane. It is also used as a raw material in the manufacture of carbon tetrachloride; as a solvent for waxes, resins, lipids and rubber; and as a perservative for fresh fruit (Klaassen et al., 1986). Other applications include use as a disinfectant, a soil fumigant, an insecticide and in the production of semiconductors.

Carbon disulfide is a clear, colorless liquid. In its pure state it smells like ether but most commercial grades have a foul odor similar to that of decaying cabbage (Gosselin et

al., 1984). Carbon disulfide is highly flammable and can easily be ignited by sparks and hot surfaces. At high temperatures carbon disulfide can react with water vapor to form hydrogen sulfide (Grayson et al., 1981)

Carbon disulfide is hazardous as a liquid and a vapor. Inhalation is the major route of toxic exposure, but this may be compounded by percutaneous absorption and ingestion. Reports of skin contamination and ingestion are rare. The most extensive data on carbon disulfide exists on chronic poisoning by vapors. It is extremely irritating to eyes, skin and mucous membranes. Following exposure to high vapor concentrations, effects on eyesight (a gradual increase in the sensitivity to light) have been observed before other symptoms became evident (Grayson et al., 1981). Because carbon disulfide can dissolve fat, it can extract it from the skin causing dryness and cracking. Carbon disulfide is reported to be one of the strongest skin irritants known, causing third degree burns within minutes (Spyker et al., 1982). Skin absorption may cause localized degeneration of peripheral nerves, usually in the hands (Sittig, 1985).

Inhalation of carbon disulfide vapor may also cause respiratory irritation and progress to bronchitis and emphysema. Carbon disulfide can be detected by odor at a concentraion of 1 ppm but the olfactory responses fatigue rapidly; thus, odor does not serve as a good warning property (Sittig, 1985).

The primary manifestations of carbon disulfide exposure include physiological, neurological and cardiovascular disorders. Acute exposures may result in blurred vision, nausea, vomiting, headache and respiratory paralysis. Delayed effects are irritability, muscle spasms, visual disturbances and psychotic behavior (Hazardline, 1987). Ingestion may cause vomiting, headache, cyanosis, peripheral vascular collapse, hypothermia, convulsions, coma. Death will occur within a few hours due to respiratory paralysis (Davidson and Feinlab, 1972). Chronic exposures have caused insomnia, nightmares,

loss of memory and impotency (Sittig, 1985). It also produces headache, dizziness and diminished motor ability with staggering gait and loss of coordination.

Carbon disulfide intoxication can involve all areas of the central and peripheral nervous systems. It produces damage to the cranial nerves, caudate nucleus and the putamen of the central nervous system. In the peripheral nerves, carbon disulfide causes axonal degeneration which may be delayed in producing symptoms. This peripheral neuropathy, called Wallarian degeneration, is generally mild compared to the central nervous system effects. It usually progresses from muscle cramps in the legs to muscle pain and parasthesis then continuing on to muscle weakness in the extremities (Klaassen et al., 1986). Parkansonian-like tremors have also been noted. Wallarian degeneration has been reported in central and peripheral nervous system fibers of rats chronically exposed to carbon disulfide (Klaassen et al., 1986). A study by Ehrhardt (1967) revealed that women are more sensitive than men to the neurotoxic effects of this compound.

Aside from the neurological disease caused from carbon disulfide exposure, derangements in other organs or systems have also been noted. Carbon disulfide has been found to be a contributing factor in coronary heart disease (Tiller et al., 1968; Hernberg et al)., 1970). Recent epidemiological studies of viscose rayon workers have shown a two- to five- fold increase in the risk of coronary heart disease, as compared to workers not exposed to carbon disulfide (Tolonem, 1975). Liver damage, although not a prominent feature, has also been an effect. Rats surviving a single oral dose showed enlargement of the liver and depression of hepatic drug-metabolizing enzyme but not liver necrosis (Bond et al., 1969). Metabolites of carbon disulfide (dithiocarbonates) have the ability to chelate metal ions such as copper and zinc (Brieger, 1967), thus depleting trace metals necessary for proper enzyme function. In animals exposed to carbon disulfide the copper content of the brain and spinal cord was significantly lowered.

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Simultaneously, marked degenerative changes were observed in axons of the spinal cord and the Purkinje cells of the cerebrum

(Cohen et al., 1959).

Carbon disulfide has been demonstrated to produce reproductive and teratogenic effects in animals when inhaled. Bariliah et al. (1975) showed that inhalation of 10  $\text{mg/m}^3$  carbon disulfide was lethal to rat embryos before and after implantation. A dose of 2.2  $\text{g/m}^3$  for 4 hours per day proved embryotoxic if given to female rats during gestation, but had no effect on male rats (Salnikova and Chirkova, 1974). Data on mutagenicity and carcinogenicity was not available.

The Federal standard for carbon disulfide exposure in air is 20 ppm. This value is an 8 hour time weighted average (TWA). The acceptable ceiling concentration is 30 ppm with a maximum peak above this for an 8 hour workshift of 100 ppm for a maximum duration of 30 minutes (Sittig, 1985). NIOSH recommends that exposure be limited to a TWA concentration of 1 ppm. The oral acceptable intake for chronic exposure (AIC) to carbon disulfide is 1.00 x 10<sup>-1</sup> mg/kg/day (SPHEM, 1986).

## 4.1.3 Cyanide

The term cyanides encompass those inorganic or organic compounds which contain the CN group. Examples include: cyanide ions that form complexes with metals, cyanates that contain the OCN radical, alkyl cyanates that trimerize to cyanurates, nitriles, and cyanohydrins. The toxicity of many of these substances is related to subsequent release of hydrogen cyanide (HCN) or the CN radical. These components can be released as a result of photodecomposition, ionization, or dissociation (Dourdoroff et al., 1966; EPA, 1980).

Cyanides are used for a variety of applications. Cyanuric chloride-based herbicides have experienced fast growth (Kirk-Othmer, 1978). Hydrogen cyanide (compressed gas) has been used as a fumigant in ships, warehouses, and in greenhouses. Many industrial effluent wastes contain

cyano-compounds including steel, plastics, synthetic fibers, and pharmaceutical and specialty chemicals, as well as the metallurgic industries (EPA, 1980).

There are some naturally occurring substances that contain cyanide. -Amygdalin and linamarin are examples of cyanogenic glycosides found in seeds of such plants as peaches, cherries, apples, and pears and in flax and lima beans, respectively. The starchy root of the cassava plant also contains a natural source of hydrogen cyanide.

The ambient water quality criterion calculated to protect freshwater aquatic organisms is 3.5 µg/l as a 24-hour average with a ceiling limit of 52  $\mu$ g/l which should not be exceeded at any time (EPA, 1980). Acute saltwater aquatic toxicity values have been reported as low as 30  $\mu$ g/l in the copopod (Acartia clausl) (EPA, 1980). The most acute freshwater invertebrate  $LC^{50}$  values range from 50  $\mu$ g/l to 200  $\mu$ g/l according to the EPA review (1980). These species were found to be more tolerant of cyanide than the freshwater fish species. The observed <u>Salvelinus</u> LC for <u>fontinalis</u> (brook trout) was 52 µg/l. Test results for two species found at the site, Lepomis macrochirús (blue gill) and Micropterus salmoides (large mouth bass) produced LC<sup>50</sup> values ranging from  $74 - 364 \mu g/l$  with juveniles being more sensitive than adults (Smith et al. 1978 and 1979). Chronic toxicity studies suggest serious reductions in longevity and growth in fish at free cyanide concentrations ranging from 20 to 50 µg/l (EPA, 1980. The Freshwater Final Chronic Value for free cyanide is 3.5  $\mu$ g/1.

Cyanides are readily absorbed through the lungs, gastrointestinal tract and skin. Death from acute cyanide poisoning is the result of "cytotoxic anoxia", or cellular asphyxiation. It is one of the most rapidly acting toxins known to man (Gilman et al., 1980). Cyanide interferes with the iron component of cytochrome oxidase, a crucial terminal enzyme in the electron transport system.

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The detoxification of cyanide in humans is extremely efficient (Klaassen et al., 1986). The extrapolated human detoxification rate has been calculated to be 0.017 mg/kg/minute (EPA, 1985). As a result of the effective detoxification mechanism, chronic toxic effects of cyanides are rare. Many chronic studies have been performed in both rodents and dogs - all with negative findings (EPA, 1980; EPA, 1985). There do not appear to be any adverse health effects in rats resulting from long-term (2 years) low dose (76-190 mg/kg) cyanide exposure (Howard and Hanzal, 1955).

There are conflicting data regarding the teratogenicity of cyanides. Significant teratogenic effects were observed in Golden Syrian hamsters in all concentration groups (78.5, 79.4, and 80.7 mg CN /kg body weight/day). High incidences of fetal resorption and fetal abnormalities were observed (Doherty. et al., 1982). Tewe and Maner (1981a) designed a dietary experiment to study the effects of KCN on the reproductive performance of female Wistar rats. Animals were fed a low dose cassava meal (21 mg HCN/kg) before, during, and after pregnancy. No significant differences were observed between treated and control groups. A similar study (Tewe and Maner, 1981b) that used pigs revealed similar negative results with the exception of significant differences found in fetal spleen-to-body and fetal heart-to-body ratios of the high-cyanide group (520.7 mg/kg diet).

Noncarcinogenic effects have been quantified by the EPA (1985). The ten-day health advisory for a 10 kilogram child, drinking 1 liter of water per day is 0.16 µg/l. An uncertainty factor of 500 was used instead of the usual factor of 100 in order to account for the uncertainty involved in deriving a drinking water criterion from a dietary study. The same 10-day health advisory for an adult (weighing 70 kilograms and consuming 2 liters of water per day) is 0.560 µg CN/L. The same safety factor of 500 was applied. The lifetime health advisory is 0.750 µg/L. The acceptable daily intake (ADI) for a 70 kilogram adult was calculated to be 1500 µg/day.

Although a safety factor of 500 was also applied in this derivation, it was for different reasons. First, an uncertainty of 100 was applied based on the National Academy of Sciences (NAS)/Office of Drinking Water (ODW) guidelines to accommodate the uncertainty of extrapolating an animal no-observed-effect-level (NOEL) for purposes of a human application. Then an additional factor of 5 was used to account for the dietary study to drinking water criterion conversion. This value has been accepted by EPA as the oral acceptable intake chronic (AIC) of 2.00 x 10<sup>-2</sup> mg/kg/day. No inhalation value has been derived.

The Carcinogen Assessment Group of the EPA has not evaluated the carcinogenicity of cyanide because there is a lack of human and animal carcinogenicity data. The EPA, therefore, has designated it as a group "D" - Not Classified Chemical.

## 4.1.4 Mercury

Mercury has been used in the past for medicinal purposes: antiseptics, antisyphilitics, cathartics, and diuretics (Gosselin et al., 1984.) There are a number of occupations associated with mercury exposure, particularly through inhalation. These include mining, smelting, chloralkali production, and the manufacture of mercury-containing products such as batteries, measuring devices (thermometers) and paints. Mercury has also been used agriculturally as a seed and cereal protectant and as a fungicide.

The World Health Organization (WHO) estimates that approximately 50% of the releases of mercury to the environment may be from natural sources (25,000 - 30,000 tons per year) and 50% from anthropogenic sources (WHO, 1976). The anthropogenic sources include fossil fuel burning and smelting operations. Ambient background levels of mercury in the air in the northern hemisphere are about 2 ng/m<sup>3</sup> (EPA, 1984a). In urban/industrial areas, airborne concentrations may be 10 ng/m<sup>3</sup> or

higher. A majority of this mercury is likely to be particle-bound.

Exposure to mercury in most forms is associated with a high degree of toxicity. Elemental (metallic) mercury causes behavioral effects and other nervous system damage. Inorganic mercury salts do not generally reach the brain, but will produce kidney damage. Divalent (mercuric) mercury is substantially more toxic in this regard than the monovalent (mercurous) form. Organic mercury compounds are also toxic. The ionic forms of mercury can be methylated by microorganisms in detritus and sediments under bodies of water (Gosselin et al., 1984).

Acute mercury poisoning due to ingestion of ionizable mercurial salts begins with the corrosive nature of the compound. Cell death occurs immediately in the mouth and throat and then affects the tissues of the esophagus and stomach (Gosselin et al., 1984); pain and vomiting ensue. Death occurs within a few hours and is attributed to peripheral vascular collapse due to severe fluid and electrolyte losses (Gosselin et al., 1984). If death does not occur within a few hours, it can be delayed several days; this depends largely on the dose received. The kidneys are a target organ with tubular nephritis progressing to complete renal failure. Acute poisoning from inorganic mercury does not involve the central or peripheral nervous systems as does acute poisoning due to organic mercury or to chronic mercury (inorganic or organic).

The pharmokinetics and pharmacodynamics of mercury depends largely on its chemical form. Inhalation of elemental mercury vapor is problematic because it has such a high vapor pressure (18 mg/m<sup>3</sup> in a saturated atmosphere) (Klaassen et al., 1986). Preferential deposition occurs in the alveolar sacs based on the monoatomic state that is assumed by the vapor. The vapor is lipid-soluble, has increased retention time in the lung, and approximately 80% is absorbed by humans (Klaassen et al., 1986). This chemical form is not readily absorbed by the gastrointestinal tract. Organic mercury, however, is

efficiently absorbed by the gastrointestinal tract based on its ability to traverse biological membranes. Distribution and metabolism of mercury are also dependent upon the chemical form. Both elemental and organic mercury degrade to divalent mercury, which is more toxic. The kidney is the target organ for the elemental form, whereas the central nervous system is the target organ for organic mercury.

The Ambient Water Quality Criterion to adequately protect freshwater aquatic life is 0.000012 mg/l for total recoverable mercury (EPA, 1985). The EPA has derived this concentration value as a 24-hour average which should not exceed 0.0024 mg/l at anytime. The criterion for total recoverable mercury used to protect saltwater oganisms is 0.025 mg/l. Sensitivities range widely among the freshwater species tested.  ${\tt LC}^{50}$ values for fish decline with increasing temperature. Increased temperature increases metabolic rate and respiratory volume which accelerates mercury uptake, since gills are a primary uptake site for most aquatic organisms. Increased temperature also leads to accelerated exposure via the food chain (Sharpe, et al. 1977). Increases in, metabolic rate lead to increases in food consumption. Because of this relationship, dissolved oxygen levels can also influence mercury uptake. When oxygen tensions are low, some aquatic organisms may be forced into warmer surface waters for sufficient oxygen. There is some evidence of a synergistic toxic response in the presence of other metals such as zinc and lead (Parker, 1979). adverse effects of mercury on aquatic organisms include reproduction, growth, metabolism, osmoregulation, and others. The saltwater criterion has also been derived as a 24-hour average and should not exceed 3.7 mg/l at any time.

The bioconcentration factor (BCF) for mercury in aquatic organisms has been reviewed by the EPA (1983). BCF values for methyl mercury (organic form) have been reported as high as 8,049 in rainbow trout (Salmo gairdneri) (Reinert et al., 1974) and as high as 64,000 in fathead minnows (Pimephales promelas) (Olson et al, 1975). These BCFs indicate that mercury

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compounds may bioaccumulate in fish and other edible aquatic organisms.

Birds and mammals may also be adversely affected by mercury. Juvenile birds are generally more sensitive than adults (Hill, 1981). Sublethal effects may involve impaired reproduction, development, and growth. Effects in mammals are similar to humans including teratogenic, mutagenic, and carcinogenic effects.

Two widespread mercury poisonings associated with consuming tainted food have been reported. Methyl mercury bioaccumulated in fish of Minamata Bay in Japan after a typhoon disturbed the Bay's bottom sediment in 1953 (Matsumoto et al., 1965). Consumption of contaminated fish by residents of Niigata and Minamata Bay, Japan caused 1,200 cases of Minamata disease including more than 100 fatalities (Tsubaki and Irukayama, 1977). Because methyl mercury can readily cross the placental barrier, the fetuses of many of the pregnant women suffered teratogenic effects and even death (Matsumoto et al., 1965).

Another widespread methyl mercury poisoning occurred in Iraq when methyl-mercury-treated seed grains were used for bread flour and consumed. Clarkson et al. (1976) described 6,500 hospital admissions and 500 fatalities.

Symptoms of chronic mercury poisoning can be both neurological and psychological in nature as the central nervous system is the primary target organ. In cases of chronic exposure to organic mercury the route of entry does not influence the symptomology (Gosselin et al., 1984). Hand and finger tremors, slurred or scanning speech patterns, and drunken-stupor-like (atoxic) gait are some motor-control impairments that have been observed in chronic mercurial toxicity. Other neurological symptoms include visual disturbances. The peripheral nervous system may also be affected. A psychological syndrome known as erethism is known to occur (Gosselin et al., 1984); it is characterized by subtle or dramatic changes in behavior and personality including

depression, fearfulness, restlessness, irritability, irascibility, timidity, indecision, and early embarrassment.

Advanced cases may also experience memory loss, hallucination, and mental deterioration.

There are acceptable intakes derived for both inorganic and organic mercury and compounds. The EPA has derived the same value for acceptable intake subchronic (AIS) and chronic (AIC) of  $2.00 \times 10^{-3}$  mg/kg/day for inorganic mercury. The inhalation-based AIS and AIC are  $5.1 \times 10^{-4}$  and  $5.10 \times 10^{-5}$  mg/kg/day for inorganic mercury, respectively. The oral AIS for organic mercury is  $2.80 \times 10^{-4}$  mg/kg/day, whereas the oral AIC is  $3.00 \times 10^{-4}$ . The inhalation AIS and AIC for organic mercury are both  $1.00 \times 10^{-4}$  mg/kg/day.

In a review of carcinogenic data for either inorganic mercury or methyl mercury, the EPA (1984) noted that none of the available data indicated "carcinogenic potential."

## 4.1.5 Thiocarbamates

The carbamates are a class of compounds with multiple uses. The biological activity and toxicity of these compounds varies greatly and is contingent on structure. Depending on the arrangement of the substitutions, the carbamates can be used as herbicides, insecticides, medicinals, nematicides or molluscicides (Grayson et al., 1981).

The thiocarbamates are a group of carbamates which contain a sulfur atom. Butylate, cycloate, EPTC, molinate, pebulate and vernolate are included in this group. They are used as herbicides and, unlike the carbamates used as insecticides, do not possess strong anticholinesterase activity (Klaassen et al., 1986).

Thiocarbamates are generally applied to the soil surface where they are rapidly absorbed by plants. It is thought that they exert their effect by interfering with enzymatic mechanisms by chelation of a metal (Grayson et al., 1981) and, therefore, inhibit photosynthesis. Because photosynthesis does

not occur in mammals, these compounds present little toxic hazard.

The acute oral LD50s in rats, for the compounds mentioned above, range from 500 mg/kg to 4000 mg/kg. The differences in these values may be due to the extent the compounds inhibit enzyme cholinesterases. Those with low LD50's can cause greater inhibition whereas this activity is too weak or metabolism is too rapid for compounds with higher values.

Butylate is used to prevent germination of grass and weeds in maize fields. The acute oral LD50 in the rat ranges from 4000-5000 mg/kg (Gosselin et al., 1984). A chronic feeding study (2 years) in rats done by Stauffer Chemical Company resulted in decreased weight gain, increased food consumption, increased liver weights and increased hepatocellular hypertrophy. At the low dose of 50 mg/kg day no effects were observed. Teratology studies in mice and rats with low doses of 4 mg/kg/day and 40 mg/kg/day, respectively, showed no deleterious effects.

Cycloate is a selective, pre-emergence herbicide used to control grassy weeds on row, crops. It has an acute oral LD50 in the rat of 2000-3000 mg/kg. Results from a subchronic inhalation study (Stauffer) in the rat showed an increase incidence in Wallerian degeneration of the peripheral nerves at 119 mg/m<sup>3</sup>. The rats were exposed for 6 hours/day, 5 days/week for 3 months. Low-dosed rats showed no histopathological effects of the peripheral nerves. A neurotoxicity study in the hen was then performed using very high doses (10,170 mg/kg - 2 doses, and 3,051 mg/kg-10 doses) and no evidence of muscle or nerve degeneration was observed. In a rat teratology study no signs of embryofetotoxicity or teratogenic effects were observed. Cycloate is irritating to the eyes and skin (Gosselin et al., 1984).

EPTC. also called eptam, is used in controlling grassy and broadleaf weeds. The acute oral LD50 in the rat is 1630 mg/kg (Gosselin et al., 1984). It is capable of inhibiting enzyme cholinesterase. In a chronic rat feeding study of two years (Stauffer) signs of toxicity included: mortality, coagulopathy

and altered carriage of the testes in males at the high dose of 125 mg/kg/day; cataracts in females at the same dose; a decrease in food consumption in both sexes at the high dose and at 25 mg/kg/day; and hind limb weakness, muscular atrophy, hind limb peripheral nerve and spinal cord axonal degeneration in both sexes at the mid and high dosage. At the low dose, 5 mg/kg/day, mild heart muscle degeneration was observed. No teratogenic effects were observed at any dosage level in a rat teratology study. EPTC is a skin and eye irritant (Gosselin et al., 1984).

Molinate is used on rice for weed control. Its acute oral LD50 in rats is 500 mg/kg (Gosselin et al., 1984). Signs of toxicity in rats fed molinate showed a decreased body weight gain, increased testicular weight and possibly decreased kidney weight. In this chronic study Fischer rats were fed 0, 8, 16 and 32 mg/kg/day for the first 18 weeks, and 0, 0.63, 2.0 and 6.32 mg/kg/day for the remaining 86 weeks of the experiment (Stauffer Chemical Company). Genetic toxicity studies using Bacillus subtilis, salmonella typhimurium and Eschericha coli were all negative as was the neurotoxicity study in hens. (These studies were also performed by Stauffer Chemical). None of the molinate treated hens exhibited signs of delayed neurotoxicity similar to those seen after administration of a positive control. Molinate produces skin and eye irritation (Gosselin et al., 1984).

Pebulate, also a grass and broadleaf herbicide, has an acute oral LD50 in rats of 1100 mg/kg. Chronic feeding studies (2 years) in the mouse (performed by Stauffer) showed decreases in erythrocyte count, hemoglobin and hematocrit, and an increase in liver weight. A subchronic inhalation study in the rat resulted in inhibition of brain and erythrocyte chloinesterase levels, kidney damage and changes in nasal epithelium. Effects were observed at 16.1 and 79.3 mg/m<sup>3</sup> (6 hours/day, 5 days/week for 14 days). Cows given repeated doses of 100 mg/kg died after 6 days (Gosselin et al., 1984). Congestion of thyroid, spleen, adrenal and lung paranchyma,

acute toxic tubular nephritis with hemorrhaging, and coagulative necrosis of the liver were discovered upon examination of the animals. Pebulate is irritating to the eyes. Skin irritation was not significant (Gosselin et al., 1984).

Vernolate is a selective thiocarbamate herbicide used on sweet potatoes, peanuts and soybeans. Its acute oral LD50 in the rat is 1500 mg/kg (Gosselin et al., 1984). Subchronic feeding studies performed by Stauffer Chemical Company showed no signs of toxicity in rats fed doses up to 32 mg/kg/day for 14 weeks or in dogs fed a high dose of 1800 ppm. Vernolate does not produce skin irritation but is irritating to the eyes (Gosselin et al., 1984).

Thiocarbamate herbicides are detoxified by enzymatic oxidation in the liver to sulfoxides. Sulfoxides do not react . significantly with cholinesterase but do combine with the sulfhydryl groups of glutathione. They are excreted as mercaptides in the urine (Gosselin et al., 1984).

## 4.1.6 Thiocyanate

Aliphatic thiocyanates have been used as contact insecticides since the 1930's. Short chain derivatives (those with low molecular weights) are volatile liquids and can be used as insecticidal fumigants. Long chain derivatives are oily liquids. These are made into dusting powders and kerosene based sprays for use in fields, gardens and homes. They have also been used dermally as delousing preparations and as livestock sprays (Gosselin et al., 1984).

The toxicity of these organic thiocyanates appears to be dependent on the length of the aliphatic side chain. Methyl, ethyl and isopropyl thiocyanates and Lethane 384 are potent and rapidly acting poisons (Gosselin et al, 1984). The mean lethal dose of methyl thiocyanate fed to rats is less than 20 mg/kg (von Oettingen et al., 1936). Effects produced by these compounds include central nervous system depression with a

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transient period of respiratory stimulation. This may progress to death due to respiratory failure (Gosselin et al., 1984). These compounds can also cause tremors, paralysis and spasticity. Opisthotonos has also been described in rabbits given Lethane 384 by various routes (Main and Haag, 1942). It is known that liver enzymes liberate cyanide from these thiocynates (von Oettingen et al., 1936), and this is probably the cause of poisoning. Butyl thiocyanate (and higher homologues) do not liberate significant amounts of cyanide in vivo (Ohkawa et al., 1972; von Oettingen et al., 1939). The mechanism for toxicity is not known for the higher thiocyanates. All thiocyanate derivatives can cause primary irritation to the skin and eyes. Undiluted solutions may produce severe cutaneous reactions (Cameron et al., 1939).

A toxicological connection between the aliphatic thiocyanates and the thiocyanate salts has not been demonstrated. The probable lethal dose for the salts (ammonium, potassium, sodium) in man is 15-30 grams if ingested all at once (Gosselin et al., 1984). Acute fatalities have occurred within 10 to 48 hours. Large overdoses induce vomitting and hyperexcitability, extreme cerebral excitement, delerium, convulsions and spasticity of extensor muscles (Gosselin et al., 1984). In chronic poisonings, symptoms include coryza, skin rashes, weakness, fatigue, vertigo, nausea, vomiting, diarrhea, confusion and aphasias (Gosselin et al., 1984). The thiocyanate ion is slowly excreted in urine. Decomposition to cyanide is not significant.

### 4.2 Standards Comparison

Following determination of exposure-point concentrations, these values must be compared to Applicable or Relevant and Appropriate Requirements (ARARs) and other federal and state criteria. These ARARs are:

Safe Drinking Water Act Maximum Contaminant Levels
 (MCLs) and Maximum Contaminant Level Goals (MCLGs)

 EPA Ambient Water Quality Criteria (both Drinking Water and Aquatic Habitat Criteria, and Drinking Water Only criteria)

## Other federal and state criteria include:

- EPA Drinking Water Health Advisories
- EPA proposed MCLs and MCLGs

These criteria are presented in Table 6-2. ARARs and other federal and state criteria for drinking water are listed in Tables 4-1 through 4-3.

## INDICATOR CHEMICALS AT THE COLD CREEK/LEMOYNE SITE,

## MOBILE COUNTY, ALABAMA

	EPA Ambient Fre Quality Grit	Lowest Reported Toxic Concentration		
Compound	Chronic	Acute	Chronic	Acute
Carbon tetrachloride	-	-	-	35.2
Carbon Disulfide			-	-
Cyanide	$5.2 \times 10^{-3}$	0.022	-	-
Mercury	1.2 x 10 <sup>-5</sup>	2.4 x 10 <sup>-3</sup>	-	-
Thiocarbamates	-	-	•	-
EPTC	-		-	•
Cycloate	-	-	-	•
Molinate		-	-	•
Pebulate	-		•	٠
Vernolate			-	·
Butyrate			-	
Thiocyanale				

TABLE 4-2

# APPLICABLE OR RELEVENT AND APPROPRIATE REQUIREMENTS (ARAR'S) FOR INDICATOR CHEMICALS AT THE COLDCREEK/LEMOYNE SITE IN MOBILE COUNTY, ALABAMA

	(mg/1) Safe Drinking Water Act MCL
Carbon Tetrachloride*	5.0 E-03
Carbon Disulfide	400 400
Cyanide	7.0 E-01
Mercury	2.0 2-03
Thiocarbamates	
EPTC	
Cycloate	
Molinate	, <del></del>
Pebulate	
Vernolate	
Butylate	NO 100
Thiocyanate	

\*Value given for potential carcinogens at a risk of  $10^{-6}$ .

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TABLE 4-3
OTHER FROERAL AND STATE CRITERIA FOR INDICATOR
CHEMICALS AT THE COLD CREEK/LEMOYNE SITE
IN MOBILE COUNTY, ALABAMA

	(mg/l)	RPA Drink	(mg/l) BPA Ambient Water	
	Reference	Health Advis		
	Dose Value	Subchronic	Chronic	Quality Criteria
		10-day 10kg	1.1fetime 70 kg	(Drinking Water)**
Carbon Tetrachloride		0.160		0.00042***
Carbon Disulfide	3.50			
Cyanide	0.7	0.00016	0.00075	2.00
Mercury	0.07		0.0011	0.01
Thiocarbamates			<b></b>	
EPTC	0.88	par - 140	•	0.00000088
Cycloate	0.05*	· •••		19.0
Molinate	0.20*	<del>-</del> -		0.0028
Pebulate	4.0*			0.002
Vernolate	0.035	<b>~</b> ~		5.0
Butylate	2.45			
Thiocyanate		<b>∞</b> .∞		

These values were provided by Stauffer from the Remedial Investigation.

<sup>\*\*</sup> Adapted from criteria for drinking water and fish consumption by U.S. EPA (U.S. EPA, 1986).

#### 5. EXPOSURE ASSESSMENT

## 5.1 Land Use and Demography

As discussed in Section 2, the Cold Creek/LeMoyne site is located approximately 20 miles north of Mobile, Alabama, and about seven miles north of Interstate Highway 65. The site is located in an industrial area and is surrounded by several other chemical production companies (Stauffer, 1987).

Based on a review of information contained in the draft Remedial Investigation (Stauffer, 1987) it appears that only a few (less than 10) residences are located within one mile of the site, and they are not downgradient of the carbon tetrachloride and carbon disulfide groundwater plumes. The largest populated areas are Mt. Vernon, approximately 8 miles. north of the site, and Creola, which is about 5 miles to the south of the site. According to the 1980 census data (U.S. Department of Commerce, 1981) the town of Creola had a population of 673 and Mt. Vérnon had a population of 1,038 in 1980.

#### 5.2 Human Health Exposure Assessment

At any waste site both humans and environmental receptors may be directly exposed to contaminants in air, water or solid media (soils, sediments or sludges) or indirectly through the food chain. The route of intake may be ingestion, inhalation or dermal absorption. Human exposure to the contaminants identified at the Cold Creek/LeMoyne site can potentially occur through the pathways discussed above. The following routes of exposure have been chosen to perform a quantitative assessment of risks because they are believed to be the most likely pathways and because empirical data is available for the different media used in these scenarios. These are:

1

Incidental ingestion of contaminated swamp sediments;

Dermal contact with contaminated swamp sediments;

Ingestion of contaminated fish; and

Ingestion of contaminated ground water.

At least two hypothetical exposure scenarios will be developed for each route of exposure. The worst case scenario will use the maximum concentration of the contaminant measured in the media (water, soil or fish), and a more realistic scenario will use the average concentration of the contaminant found in each media at the site. These two scenarios will provide a range of plausible body dose levels and help limit the uncertainties surrounding these estimates. In addition, conservative assumptions will be used for the quantity of media contacted or ingested, the percent of the contaminant absorbed (either through the skin or through the gastrointestinal tract), and the frequency and duration of exposure. These assumptions will tend to overestimate rather than underestimate the actual exposure at this site. Appendix A contains worksheets showing the dose calculations for each scenario.

## 5.2.1 Ingestion of Contaminated Swamp Sediments

Children may inadvertently ingest soil when they play outdoors and adults may also inadvertently ingest soil while fishing or engaging in outdoor activities. This exposure scenario is estimated by combining the concentration of the contaminant in soil, the rate of soil ingestion, the estimated body weight of the exposed individual, and the frequency of exposure. The equation used to calculate the exposure is given in Table 5-1.

This risk assessment assumes that exposure to contaminated swamp sediments at the site will be infrequent because the site is located in an industrial area and usually people do not

Chronic Daily

TABLE 5-1. FORMULAS	FOR CALCULATING BOD	Y DOSE LEVELS.				4
CHRONIC DAILY INTAKE	FOR INCIDENTAL SOIL	INGESTION				W
Chronic Daily Intake (mg/kg/day)	. •	Amount of soil ingested (mg/visit)	Absorption X factor	X (visits/year)	X Duration (years) X (1 kg/	rsion factor (N)
intent (mg/kg/cay)		Body Weight (kg)	X Days in a year (365 days/1 year)			01
CHRONIC DAILY INTAKE	FOR DERHAL ABSORPTI	CN				92
Chronic Daily Intake (mg/kg/day)	Concentration in Soil (mg/kg) X		Soil Concentration X on skin (mg/cm2)  Body Weight (kg)	X factor	X (visits/year) X Durat  X Lifetime *	xposure Conversion factor ion (years) X (1 kg/1,000,000 mg)
CHRONIC DAILY INTAKE	FOR INGESTION OF CO	MTAMINATED FISH				
Chronic Daily Intake (mg/kg/day) :		Amount of fish ingested (mg/day)	Absorption X factor	• •	Conversion factor X (1 kg/1,000,000 mg)	
	•	Body Weight (kg)	X Lifetime * (years)			
CHRONIC DAILY INTAKE	FOR INGESTION OF CO	INTAMINATED GROUND W	ATER			

X Duration (years)

factor

Lifetime \*

(years)

water (mg/l) X ingested (l/day) X

Intake (mg/kg/day) = -----

Body Weight (kg)

<sup>\*</sup>Years in lifetime or in the period over which risk is being etimated (70 year lifetime for carcinogens, less than lifetime for noncarcinogens).

spend a lot of time in wetland areas unless they are bird watchers or are on other types of nature walks. In addition, people are expected to be protected from direct contact with sediments by shoes and other articles of clothing.

Nonetheless, the following exposure scenarios have been developed for teenagers and for industrial workers on-site.

As noted above, the contaminated swamp sediments are located in an area surrounded by chemical manufacturing facilities; however, older children (aged 12 to 17) may on occasion gain access to the site. Although the frequency at which such visits would occur is highly uncertain, it is assumed to occur 6 times per year for a period of 5 years. The average weight of a teenager is estimated to be 50 kilograms (Anderson et al., 1985) and the absorption factor is assumed to be 1 for both inorganic and organic compounds.

Estimates of the amount of soil ingested by older children and adults are based on little direct data and vary widely. Therefore the risk assessment assumes that teenagers and adults ingest soil at the same daily rate as has been measured in young children, although the frequency is expected to be much lower. The minimum soil ingestion reported in the literature is 10 micrograms per day, based on a presumed intake of soiled candies (Day, et al., 1975), while the maximum soil ingestion has been estimated to be 10 grams per day (Kimbrough et al., 1984). The high end of the predicted soil ingestion rates has been adjusted downward by the EPA (1986a) and the EPA has also acknowledged that the high level of intake is probably only pertinent for children with pica (a syndrome in which children intentionally eat foreign objects). Recent studies using trace elements in fecal material as indicators of soil ingestion in children, indicate that the low end of the estimated range may also be incorrect. Therefore, ERT has used 100 milligrams per day based on a study of soil ingestion rates in children by Clausing et al. (1987), which is the only published and

peer-reviewed report of soil ingestion estimates using trace element techniques. Clausing et al. (1987) report that the mean daily soil ingestion by nursery school children is 100 milligrams, with a standard deviation of 67 milligrams.

On-site workers are more likely to come in contact with the contaminated swamp sediments than teenagers, although such occurrences are probably relatively infrequent. For the purpose of this assessment, it is assumed that an individual may come in contact with the sediments 12 times a year for a period of 30 years. The average body weight of an adult worker is assumed to be 70 kilograms, and the amount of ingested sediments is expected to be 100 milligrams, as discussed above.

## 5.2.2 Dermal Contact with Contaminated Swamp Sediments

When children and adults are outside and contact soil or sediments, they may also absorb some of the contaminants through the skin. Dermal exposure is estimated by combining the concentration of the contaminant in the sediment, the amount of skin exposed to the soil, the weight of the person, the percent of absorption through the skin, and the frequency of the exposure. The equation for estimating dermal exposure is shown in Table 5-1.

The assumptions regarding the frequency of exposure and the body weight of exposed individuals are the same as those used in the scenarios developed for incidental soil ingestion discussed above. For this assessment, only the hands of exposed individuals are assumed to come into contact with contaminated sediments. Teenagers' hands are assumed to have a surface area of 810 square centimeters, and adults' hands are reported to have an average surface area of 1,000 square centimeters (Anderson et al., 1985). It is also assumed that the amount of soil on the skin will be 0.5 milligrams of soil per square centimeter. This value is based on a study that used tape to remove soil from children's hands and determined that approximately 0.5 milligrams of soil per square centimeter adhered to a child's hand (Lepow et al., 1974).

Little information is available on the rate of absorption through intact skin. Inorganic compounds do not appear to pass through intact skin readily, and, in general, the EPA considers dermal exposure to inorganics to be of minor importance (EPA, 1981, 1984a, 1984b, 1984c, 1986b). Therefore, this risk assessment assumes that mercury (the only inorganic contaminant of concern) is not absorbed through the skin. Little information is available on dermal absorption of the thiocarbamate indicator compounds either; however, dermal absorption of TCDD (dioxin) in rats has been studied (Poiger and Schlatter, 1980). They applied different concentrations of TCDD contaminated oil to the skin of rats and measured the uptake in the liver, which ranged from less than 1 percent to about 3 percent. An intermediate value of 1 percent absorption is used in this risk assessment, and is applied to all organic indicator compounds.

## 5.2.3 Ingestion of Contaminated Fish

Potential exposure may result from the ingestion of contaminated biota. Exposure through this scenario is estimated by combining the same factors used for estimating the risk from the ingestion of contaminated soil (i.e., the concentration of the chemical in fish, the amount of fish ingested, the estimated body weight, and the frequency of exposure). Table 5-1 shows the equation used for estimating this risk.

Although it is unlikely that anyone fishes in Cold Creek near the site (given its proximity to industrial property, its restricted access, and the small size of the fish (Stauffer, 1987)), measurable levels of mercury have been found in whole fish caught in Cold Creek. Therefore, this endangerment assessment will assume that people could catch and eat contaminated fish from the site. The maximum and average concentration found in the five fish samples are used for the worst case and realistic scenarios, respectively, even though

these concentrations will greatly overestimate the risk from this exposure route because the homogenate samples include concentrations of mercury in nonedible portions of the fish such as the liver and the skin (which tend to store the contaminants). In addition, the use of the average and maximum levels of mercury found in fish caught Cold Creek Swamp is also representative of levels measured in fish in the Mobile River over the past 18 years. A review of fish sample data from the Alabama Water commission showed that the levels of mercury found in fish in the Mobile River just south of the confluence with Cold Creek were generally within these ranges and that elevated levels of mercury (greater than 4 ppm) have also been found in fish caught in David's Lake, which is located approximately 15 miles upstream of Cold Creek Swamp. For the purpose of this assessment it is assumed that an adult weighing 70 kilograms will consume 26 grams of fish per day (which is equivalent to about one fish meal per week) for a period of 30 years; and that a teenager weighing 50 kilograms would consume the same amount of fish for a period of five years. absorption factor used for, both scenarios is one.

## 5.2.4 Ingestion of Contaminated Ground Water

The last exposure route considered in this report is ingestion of contaminated ground water. The same factors (such as absorption, body weight, and exposure duration) used for the other ingestion scenarios (sediment and fish) are also used to quantify the exposure to ground water. A review of the data presented in the Draft Remedial Investigation (Stauffer, 1987) shows that none of the indicator compounds have been detected in any of the drinking water wells located on or in the immediate vicinity of the site. Therefore, there is no current risk associated with this exposure route.

It is highly unlikely that any new drinking water wells would be installed that might intercept the contaminated ground water because a permit must be obtained from the State of Alabama before a potable water well can be installed, and it is

improbable that the State would permit any well that would be located on or adjacent to a known Superfund site with documented groundwater contamination. As discussed in more detail in the Remedial Investigation (Stauffer, 1988), the existing groundwater treatment system appears to be very efficient in preventing contaminant migration toward any of the industrial drinking water wells. However, if such an occurrence were to happen there would still be minimal exposure (if any) because the existing water wells are closely monitored to ensure that they are remaining uncontaminated, and they would be shut down as soon as they became contaminated.

Nonetheless, it is possible, although highly unlikely, that a drinking water well could be installed that might intercept the contaminated ground water. Therefore, for the purpose of this assessment, an exposure scenario will be developed using both the maximum and average concentrations of the indicator compounds found at the Cold Creek/LeMoyne site. Both the worst case and realistic exposure scenarios assume that a 70 kilogram adult worker will drink 2 liters of water per day for 30 years. An absorption factor of one is used for both inorganics and organics.

The scenario assumes that a drinking water well is installed downgradient of the groundwater interceptor system that is currently in place. In this scenario, only data from the monitoring wells downgradient from the interceptor wells were used. Both the maximum and the average contaminant value were used for each of the indicator compounds.

## 5.3 Environmental Exposure Assessment

## 5.3.1 Ecological Setting

The upland areas of the site consist of mixed pine-oak woods typical of the southeastern United States. The dominant

canopy—is <u>Pinus palustris</u> (longleaf pine) with intermixed oaks (<u>Quercus alba</u>, <u>Quercus falcata</u>), other pines (<u>P. taeda</u>, <u>P. elliotii</u>), hickories (<u>Carya spp.</u>), and sugarberry (<u>Celtis laevigata</u>). The understory consists predominantly of canopy seedlings and saplings, flowering dogwood (<u>Cornus florida</u>), and <u>Vitis sp.</u> Herbaceous ground cover is typical for the area. The site is appropriate habitat for a variety of invertebrates, amphibians and reptiles, birds and mammals; however, an inventory of these organisms actually present at the site is not available.

At lower relief, the flood plain consists of palustrine wetland consisting of temporarily flooded bottomland hardwoods and seasonally or semi-permanently flooded needle-leaved deciduous wetlands (cypress swamps). Small lateral ponds are interspersed throughout. Canopy species typical of southern . bottomland forested wetlands are found there. Examples are numerous oaks (Quercus michauxii, Q. shumardii, Q. lyrata, and Q. laurifolia), red maple (Acer rubrum), and sweetgum (Liquidambar styraciflua). A more extensive vegetation inventory can be found in the Preliminary Natural Resource Survey (USFW, 1986). The dominant trees of the cypress swamp are bald cypress (Taxodium distichum) and water tupelo (Nyssa aquatica).

Cold Creek and an unnamed tributary flow through the swamp to the Mobile River. The water current is slow. Areas without an overstory of trees are dominated by emergent aquatic macrophytes such as Typha and Pontideria.

With the exception of fish, animals have not been sampled at the site. Observations of various birds, especially waterfowl, have been reported. The United States Fish and Wildlife Service considered the wetlands at the site to be important habitat for waterfowl, and as habitat for other migrators such as waders. A general discussion of their food resources may be found in the preliminary Natural Resource Survey (USFWS, 1986).

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Two species of special concern are also known to occur in the vicinity: the American Alligator (Alligator mississippiensis, considered to be threatened by reason of similarity) and the Alabama Red-Bellied Turtle (Pseudemys alabamensis, proposed for listing). The alligator has been reported at the site.

A variety of sport and forage fishes were collected from Cold Creek and the swamp tributary in August, 1986. species list is found in Table 5-2. These fish (except the American eel, and one 300 gram fish which were excluded) were collected for mercury body burden analysis. The majority of the fish were quite small; many were no doubt young-of-theyear. Additional species are also present in the adjoining Mobile River. Three of these are anadromous species: saxatilis (Atlantic Striped bass), Alosa alabamae (Alabama shad), and Acipenser oxyrhynchus (Atlantic sturgeon). shovelnose sturgeon, <u>Scaphirhynchus</u> platorynchus, is also potentially present. This fish is a candidate for listing as a rare or endangered species. A number of the small forage fish found in the creek may spend their entire life cycles in the swamp. The larger species, especially the centrarchids (sunfish and largemouth bass) may be more mobile, ranging into the Mobile River which reduces their exposure to contamination but increases the likelihood of their being caught and eaten by sportfishers. The majority of small fish (omnivores) have a mixed diet, but the perch, sunfish, and largemouth bass are predaceous, even as juveniles. No aquatic sampling has been conducted to characterize plankton, benthic invertebrates, amphibians, etc.

TABLE 5-2
FISH SPECIES COLLECTED FROM COLD CREEK
AND TRIBUTARY, AUGUST, 1986

Scientific Name	Common Name	Cold Creek	Tributary
Amia calva	Bowfin		x
Anguilla rostrata	American eel		x
Aphedoderus sayanus	Pirate perch		x
Erimyzon sucetta	Lake chubsucker		x
Fundulus chrysotus	Golden topminnow		x
Fundulus olivaceus	Blackspotted topminnow	x	
Ictalurus natalis	Yellow bullhead		x
Lepomis gulosus	Warmouth	x	х .
Lepomis macrochirus	Bluegill	x	×
Lepomis marginatus	Dollar sunfish	x	x
Lepomis megalotis	Longear sunfish	x	x
Lepomis microlophus	Redear sunfish	x	
Lepomis punctatus	Spotted sunfish	x	x
Micropterus salmoides	Largemouth bass	x	x
Notemigonus crysoleucas	Golden shiner	x	x

### 5.3.2 Environmental Exposure Assessment

Given the industrial nature of the upland portion of the site, the most likely and consistent routes of exposure to biota are associated with the wetlands. Even terrestrial mammals, reptiles, amphibians, or birds may be exposed via these avenues.

Exposure in the wetlands may be accomplished through four main routes:

- direct exposure to contaminated water
- direct exposure to contaminated air
- direct exposure to contaminated sediments
- consumption of contaminated food

Data are not sufficient to quantify risks associated with individual exposure routes; however, a general discussion of plausible exposure scenarios follows. Among the contaminants of concern chosen for evaluation of risk to human health, the environmental discussion will be limited to exposure and subsequent risks pertaining to exposure to mercury because:

- mercury is known to biomagnify and represents a potentially significant toxicant in the environment.
- site-specific date are available for mercury showing elevated levels in wetland sediments, water, and fish.
- ambient water quality criteria are available for mercury.

Other metals are present in the swamp sediments, but data for other media are limited. In some cases (beryllium, chromium, copper, cadmiuum, and silver) the analytical detection limits exceed ambient water quality chronic criteria or no observable effect levels. For some metals, the criteria are exceeded only at a subset of sampling points. These include copper, lead,

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and zinc. Metal concentrations in wetland surface waters are found in the Final Work Plan (RI/FS) (CDM, 1985). Mercury is the metal which is consistantly found to exceed Ambient Water Quality Criteria (both acute and chronic), even in areas of the site designated to represent background concentrations. In a qualitative discussion, the exposure to mercury can be used to represent all the sediment-bound metals. Standards and environmental evaluations of thiocarbamates are not yet available, and although ambient water quality criteria are available for cyanide (see Section 4), data to assess exposure in the Cold Creek Swamp are not. Data are not available to evaluate exposure in the air, but this is not likely to be an important exposure pathway.

### Exposure to Water

Direct exposure to mercury contaminated water is generally an important exposure pathway for aquatic organisms who cannot remove themselves from the water and semi-aquatic organisms.

Most of the absorption is through the high surface area of the gills. The accepted bioconcentration factor (for brook trout) is 23,000. Mercury BCFs for juvenile Lepomis macrochirus (bluegills) which are found at the site are lower, but some BCFs for other species are much higher.

The average total mercury concentration in the surface water is 0.022 mg/L (range 0.010-0.099) which exceed Ambient Water Quality Criteria for mercury. Estimates of fish body burdens using the formula:

# BCF = body burden ppm water concentration ppm

yield estimates several orders of magnitude higher than those found in the fish. This indicates that either the BCF is inappropriate for these species, exposure at these concentrations is not constant, or the fish which were sampled had not reached uptake/depuration equilibrium for mercury. 5-13

1303G PG-361-400

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The background values for the site also appear to be as high as many of the other sampling locations. Exposure to contaminated water may represent an important exposure route. However, some of the species of fish present in the swamp, especially the cyprinids, and centrachids are quite mobile. These fish may spend only a portion of their life cycle in the swamp: as juveniles and on a more limited basis, as spawning adults. Amphibians which breed in the swamp would also be exposed on only a limited basis, however, anuran embryos may be extremely sensitive to mercury exposure (Birge and Just, 1973).

### Exposure to Sediments

Sediment concentrations of mercury are elevated in some areas of the wetland. The extent to which this mercury is available is not known, but sulfide which is also present may bind a large portion of the mercury in relatively unavailable form to the sediments.

Sediment exposure would be greatest for those organisms which reside in or on the sediments, notably benthic invertebrates. Some of these organisms are active, and may resuspend particles (bioturbidity) to the water column. Physical mechanisms of resuspension such as wind, waves, and current flow, would be minimal. Other organisms spend only a portion of their time in direct contact with sediments. Examples are:

- Aquatic insects whose life cycle includes a larval form which is associated with sediments.
- Benthic feeding fish, crayfish or other organisms.
   The primary food source may be benthic animals or plants but sediment contact and ingestion could be substantial.

- Nest-building fish, such as centrarchids and pirate perch, whose eggs are laid in the sediment surface after it is first cleared of organic debris by the activity of the adults.
- Wading birds and mammals such as muskrat and beaver.

### Exposure via the Food Web

Mercury bioconcentrates readily in many freshwater aquatic organisms, and depuration rates are slow. At higher trophic levels the most significant mercury exposure route is likely to be food. Mercury concentrates in the tissues of some aquatic plants, which may be potential food sources for crayfish, ducks, and other herbivores and omnivores at the site.

Body burden data are available only for whole body composite samples of fish. The FDA action level of 1.0 mg/kg was exceeded at two sampling sites. There are a number of problems associated with the interpretation of these values. The composites were made up of a variety of species representing different trophic levels. Many of the fish were juveniles, so an unknown but potentially large portion of the measured mercury may not have been assimilated. Other data such as fillet concentration and metal speciation are not available. This restricts our ability to assess the contribution of various exposure routes to the fish, or to estimate body burdens of their food resources. Additionally, there has been an historical problem concerning mercury contamination of fish in the Mobile and other area rivers. Samples taken over the past decade in the area of Cold Creek do suggest bioaccumulation with mercury levels in largemouth bass generally higher than in bluegills. However, the source of this contamination is not known.

In any case, the fish samples do have levels of contamination which represent a potentially harmful source for higher trophic levels. These include the alligator, other

### 3 12 0205

larger fish, fish-eating birds, and mammals such as raccoons and minks. To protect sensitive species of birds and mammals that consume fish as a major portion of their diet, suggested criteria for mercury levels in food items are contained in Table 5-3. The formula for chronic daily intake for ingestion of contaminated fish (Table 5-1) can be used to estimate daily intake for birds and mammals and compare these to the proposed criteria. A worst case scenario would assume that a 1.5 kg resident bird consumes maximally contaminated fish at a rate of 0.15 kg/day for a five year lifetime. The daily dose is 0.310 mg/kg which is less than the literature values of concern, although the diet values are exceeded. Similarly, for an 8 kg resident mammal consuming maximally contaminated fish at a rate of 0.8 kg/day for a five year lifetime, the daily does per kg body weight is the same, but this exceeds the recommended daily dose maximum for mammals.

# TABLE 5-3 PROPOSED MERCURY CRITERIA FOR PROTECTION OF SENSITIVE BIRDS AND MAMMALS\*

	Hg Concentration	Reference
Bird		
Diet (µg/kg fresh weight)	50 to <100	Heinz 1979;
		March et al 1983
Daily dose (µg/kg body weight)	<640	Spann et al. 1972;
		McEwen et al. 1973;
		Mullins et al. 1977
Mammals		
Diet (µg/kg fresh weight)	<1,100	Kucera 1979
Daily dose (µg/kg body weight)	<250	Ramprashad and
		Ronald, 1977;
		Ronald et al, 1977;
,		Khera, 1979.

<sup>\*</sup>After Eisler, 1987.

### 6. RISK CHARACTERIZATION

This section quantifies the potential for adverse health effects due to site-related chemical exposure. Because noncarcinogenic effects are assumed to have a threshold dose below which an adverse health effect will not occur, and carcinogenic effects are assumed not to have a threshold dose, risk estimates for adverse noncarcinogenic effects are determined separately from carcinogenic risks.

### 6.1 Noncarcinogenic Risk

Representative and maximum intakes of indicator chemicals that may lead to noncarcinogenic effects are compared to acceptable daily intake values. Because compounds within a chemical class may have different toxicological effects, these values are compound specific and cannot be derived for a general class. Reference doses for chronic exposure to carbon disulfide, carbon tetrachloride, cyanide, alkyl and inorganic mercury and some of the thiocarbamates, have been developed by the EPA from animal and epidemiological chemical exposure studies. Reference doses for cycloate and pebulate have not been developed by the EPA at this time, but have been calculated by ERT for this endangerment assessment based on the no-observed-effect-levels (NOELs). Thiocyanates were analyzed as total thiocyanate, therefore, the distribution of the specific compounds was undetermined and acceptable daily intake values could not be calculated.

NOELs are the highest dose administered that did not produce toxic effects in laboratory animals. NOELs for the thiocarbamates have been provided by Stauffer Chemical in the Remedial Investigation (Stauffer, 1987). To determine a reference dose in humans the NOEL in animals is multiplied by the body weight of an average human (70 kg; SPHEM, 1986) then divided by an uncertainty factor. An uncertainty factor of 100 was employed in these calculations: 10 for extrapolation from

laboratory animals to humans; and 10 for the variability between humans. For chemicals without good chronic exposure data, an uncertainty factor of 1000 was used. The Reference doses are given in Tables 6-1 and 6-2.

The potential for noncarcinogenic health effects is assessed by dividing each indicator chemical's exposure-route and duration-specific intake by the Reference Dose. This ratio or comparison is called the Hazard Index (HI). estimated intake (in mg/kg/day) is greater than the reference dose (in mg/kg/day), the HI will exceed one (unity), indicating the potential for an adverse health effect exists. An HI of less than unity occurs when the estimated intake does not exceed the acceptable intake, and indicates that levels of intake are lower than those expected to produce toxic effects. Therefore, the potential for an adverse health effect does not exist. The sum of the HIs for all exposure routes for each chemical represents the chemical-specific HI. The sum of all chemical-specific HIs gives the total exposure-point specific However, care must be taken when interpreting summed HIs, because by adding them, it is assumed that their toxicological endpoints are the same and their toxicological effects, therefore, are additive. This is not true for all chemicals. For example, some chemicals will affect the liver while others will target the kidneys or the central nervous system.

Chronic daily intakes were calculated for the indicator compounds for each of the exposure scenarios described in Section 5. The HI for each compound and each scenario are presented in Tables 6-1 and 6-2. These tables also show the sum of all exposures (and risks) at the site.

As shown in Table 6-1, adult workers who come in contact with contaminated sediments, drink water from wells located downgradient of the groundwater treatment system, and eat fish from Cold Creek swamp are not at risk. The total HI for worst case and realistic exposures fall below unity.

Table 6-1. Chronic Mazard Index (noncarcinogens) for Adult Workers at the Cold Creek/LeNoyne Sites

	-		Ground Water	į	Ingestion of	Ingestion of Swamp Sediments				
	Daily Intake (mg/kg/day) Worst Case Realistic Case	(mg/kg/day) alistic Case		Mazard index Worst Case Realistic Case	Daily Intake Worst Case Re	Daily Intake (mg/kg/day) Worst Case Realistic Case	Mazard Index Worst Case Realistic Case	dex alistic Case		
Compound (mg/kg/dmy)										
	2.75E-03	4.07E-04								
1.00£-01	5.566-04	3.67E-05	5.566-03	3.676-04	0.00€+00	0.00€+00	0.00€+00	0.00€+00		
2.00E-02										
2.00E-03	0.00€+00	0.00€+00	0.00€+00	0.00€+00	1.396-05	9.206-07	6.95E-03	4.60E-04		
2.50€-02	2.57E-04	1.96E-05	1.03E-02	7.846-04	2.01E-06	3.596-09	8.04E-07	1.446-07		
7.00E-02	8.336-05	6.12E-06	1.196-03	8.75E-05	3.62E-06	6.04E-09	5.17E-07	8.636-08		
1.006-03	9.186-05	6.12E-06	9.186-02	6.12E-03	2.21E-08	3.746-09	2.216-05	3.746-06		
1.60€-02	0.00E+00	0.00€+00	0.00€+00	0.00€+00	6.04E-09	8.648-10	3.778-07	5.406-08	•	
2.00E-03	3.166-04	3.55E-05	1.596-01	1.78€-02	1.816-08	3.886-09	9.05E-06	1.946-06		
5.006-03	6.98E-05	4.906-06	1.406-02	9.806-04	3.62E-08	7.196-09	7.248-06	1.446-06		
			2.62E-01	2.616-02			6.996-03	4.67E-04		
	Derme	Dermel Absorption of Summy Sediments	of Swamp Sedi	iments		5	Consumption			
Reference	Daily Intake (mg/kg/dey)	(mg/kg/day)	Hazard Index	Index	Daily Intake		Hazard Index	× × × ×	Total #	Total Mazard Index
Dose	Morst Case Realistic Case	alistic Case	Worst Case I	Worst Case Realistic Case	Worst Case Re	Worst Case Resistic Case	Worst Case Realistic Case	elistic Case	Worst Case A	Worst Case Realistic Case
Compound (mg/kg/dey)										
1.006-01	0.00E+00	0.00€+00	0.00€+00	0.00€+00					5.56E-03	3.67E-04
2.006-02										
2.00E-03	0.00€+00	0.00€+00	0.00E+00	0.00€+00	4.936-04	1.965-04	2.46E-01	9.80E-02	2.53E-01	9.85E-02
2.506-02	5.036-10	8.996-11	2.01E-08	3.606-09					1.196-03	8.766-05
7.00€-02	9.066-10	1.516-10	1.296-08	2.166-09					9.18E-02	6.128-03
1.00E-03	5.546-10	9.346-11	5.54E-07	9.346-08					2.27E-05	3.836-06
1.60E-02	1.51E-10	2.16E-11	9.44E-09	1.356-09					1.596-01	1.786-02
2.00E-03	4.53E-10	9.71E-11	2.26E-07	4.86E-08					1.40E-02	9.82E-04
5.00E-03	9.06E-10	1.806-10	1.816-07	3.606-08					7.42E-06	1.478-06
					•					
			1.00E-06	1.85E-07			2.46£-01	9.80€-02	5.256-01	1.246-01

S

TABLE 6-2. Chronic Hazard Index (noncarcinogens) for Teenagers at the Cold Creek/LeMoyne Site.

liments	Ders	mol Absorption	of Suamp Sedii	
rd Index	Daile tare	re (mg/kg/day)		
se Realistic Case	Worst Case R	Realistic Case	Hezerd II Worst Case R	ndex nolicaio com
			moret case ki	POLISTIC CAS
04 5.35E-05				
04 5.35E-05	0.00E+00	0.00€+00	0.00E+00	0.00E+00
08 1 ARE-DR				
7.502.00	9.51E-11	1.70E-11	3.80E-09	6.80E-10
	1.71E-10	2.85E-11	2.44E-09	4.07E-10
**************************************	1.05E-10	1.77E-11	1.05E-07	1.77E-08
( 0.2.2 0)	2.85E-11	4.08E-12	1.78E-09	2.55E-10
6 2.26E-07	8.56E-11	1.83E-11	4.28E-06	9.15E-09
1.68È-07	1.71E-10	3.40E-11	3.42E-08	6.80E-09
4 5.44F-05				
4 5.44E-05			1.90E-07	3.50E-08
n				
d Index	Total Ha	zard Index		
• Realistic Case	Worst Case Re			
2.296-02	5.83E-02	3 70		
	J. 03E-02	2.30E-02		
	9.78E-08	4 7/2 45		
	6.29E-08	1.74E-08		
	2.69E-06	1.05E-08		
		4.54E-07		
	4.58E-08	6.57E-09		
	1.10E-06	2.36E-07		
	8.80E-07	1.75E-07		
		•		

Finally, the hazard indices calculated using scenarios where teenagers are exposed to contaminated swamp sediments and consume contaminated fish are shown in Table 6-2. The total hazard index for all routes of exposure and all contaminants is very small for both worst case exposures and realistic exposures  $(5.83 \times 10^{-2})$  and  $(5.83 \times 10^{-2})$  and  $(5.83 \times 10^{-2})$  respectively).

### 6.2 Carcinogenic Risk

The underlying assumption for assessing the risks from suspect or known human carcinogens is that there is no non-zero dose that is without some finite level of risk. Carcinogenic risk is quantified by multiplying an EPA-derived cancer potency factor (the upper 95% confidence probability of cancer per unit dose of a chemical, based on dose-response data, over a lifetime) by the estimated intake (dose) to calculate cancer risk due to representative and for all maximum carcinogenic intakes represents the estimated upper range of total excess cancer risk for individuals assumed to have those site-related exposures.

The only indicator compound at the Cold Creek/LeMoyne site which is considered by the EPA to be a suspect human carcinogen is carbon tetrachloride. This compound was only detected in the groundwater and in subsurface soil samples from beneath the waste water treatment ponds, therefore, there is no current risk from carbon tetrachloride at this site. As discussed earlier, however, there is a remote possibility that groundwater could become a drinking water source in the future. In such a case, the carcinogenic risk from carbon tetrachloride (as shown in the data worksheets in Appendix A) ranges from 3.57 x 10<sup>-4</sup> for the worst possible case (i.e., if a worker ingests water having the maximum concentration of carbon tetrachloride at a rate of 2 liters per day for 30 years) to 5.28 x 10<sup>-5</sup> (based on the average concentration found in monitoring wells down gradient of the groundwater

1

interceptor system). These risks correspond to an excess chance of getting cancer of about 3 in 10,000 and 5 in 100,000 respectively.

#### 6.3 Environmental Risk

The characterization of environmental risk depends on the individuals and populations exposed and the exposure(s) to which they are subjected, plus a careful examination of the ecosystem interactions which may be altered by subtle changes in population dynamics. At the site, the most likely area of concern is the Cold Creek swamp where sediments, water, and fish have elevated mercury levels. Data are not available to quantify exposure via water, sediments, or food consumption, although given the levels of mercury in the fish, all of these exposure mechanisms are probably operating to one extent or another.

There is the potential for sensitive birds, mammals, and other organisms in residence to be affected by consuming contaminated fish along with other exposures. Although a worst-case scenario was used to estimate the potential for harm, an uncertainty factor to compensate for extrapolation from non site specific species should also be employed (see Section 6.1). This is a standard procedure used to help insure a protective margin for sensitive species, since the majority have not been clinically evaluated for mercury effects. Within this framework, recommended maximum daily doses of mercury could be exceeded by some birds and mammals. Due to the mobility of these organisms, it is unlikely that even resident species would feed full time at the site. Many of the birds are migratory, and would only be exposed for a short length of time. However, mercury does bioaccumulate. Extrapolating such an approach to the potentially threatened or endangered species which may inhabit the area would be inappropriate given the lack of site data and mercury-related reptilian studies.

### 3 12 0213

Changes in population dynamics of plants or animals, relative abundances, and diversity have implications for overall ecosystems productivity, function, and structure. However, without an assessment of the biota at the site, and establishment of actual or potential effects due to mercury contamination, the potential for ecosystem effects cannot be evaluated.

# 3 12 0214 7. SOURCES OF UNCERTAINTY

The process of an endangerment assessment usually involves several general steps: identification and selection of indicator compounds; quantification of their exposures; evaluation of the toxicity of the indicator chemicals; and the prediction of risk from these chemicals. Within any of these steps, numerous assumptions must be made. Some of the assumptions have a great deal of scientific basis, others have very little. Because we are not absolutely certain about any of the assumptions, some level of uncertainty is introduced into the endangerment assessment every time an assumption is made. In this section, some of the assumptions that introduce the greatest amounts of uncertainty and their effect on the estimates of risk are discussed. The discussion of their effect will be qualitative, not quantitative, because in most instances there is not enough information to quantify how large or small the uncertainties may be. The section is divided into subsections that correspond to the four steps involved in the risk assessment process.

### 7.1 Hazard Identification and Indicator-Compound Selection

During this step of an endangerment assessment, information on the types, concentrations, distribution and frequency of chemicals on the site is combined with measures of the toxicity of each of those chemicals to determine their potential risk. In determining the potential risk, uncertainty can be introduced in several places. Some of the more important sources of uncertainty are presented below.

Sampling error. The sampling locations at any site may not identify all the compounds on, and contaminated areas of, the site. In most cases, enough sampling has taken place so that all widely distributed compounds and highly contaminated areas are accounted for in the endangerment assessment. This source of uncertainty should make a small contribution to the total uncertainty.

- Measurement error. Numerous and complicated analyses that require a great deal of manipulation are needed to identify and quantify the compounds present on a hazardous waste site. The quantity of compounds present on the site can be either underestimated or overestimated. On rare occasions, compounds may also be misidentified. This source of uncertainty should contribute only a small amount because many samples are taken in duplicate, and laboratories have Quality Assurance/Quality Control (QA/QC) programs.
- Toxicity. Initial evaluation of a compound is dependent, in part, upon the toxicity of the compound. Some compounds have not been investigated in sufficient detail to insure that all of their toxic properties or that the severity of their toxic effects are well quantified. It is likely, however, that the most toxic compounds have been identified and that this source of uncertainty contributes little to the overall uncertainty of the risk assessment.

## 7.2 Estimation of Exposure-Point Concentrations and Human Exposure

During this step of the endangerment assessment, the concentration of indicator chemicals is either measured or estimated in the media with which human or environmental receptors will come into contact. In many cases, contaminant levels in media that may be contacted by a receptor cannot, or have not, been measured directly. In such cases, concentrations need to be estimated or modeled. Estimates and models require assumptions and these lead to uncertainty. Once the concentration in a medium is known, human exposure and dose

need to be estimated. These, too, require assumptions that lead to uncertainty. The more important sources of uncertainty are discussed below.

- Modeling assumptions. Because the baseline risk assessment examines risk over the next 70 years, concentrations of contaminants in the future need to be estimated. In most cases simplistic assumptions are employed in the models used to estimate future concentrations. These almost always result in an overestimation of future exposure and risk.

  Depending upon how simple the assumptions were, the overestimation can be quite large. The intent is to err on the side of public health; therefore, the models and assumptions are unlikely to underestimate future exposure and risk. This source of uncertainty can make an important contribution to the overall uncertainty of the risk assessment.
- Non-quantifiable exposure routes. In some cases there may not be enough information available to quantitatively estimate the magnitude of some current and future pathways of exposure. In these instances, potential exposures must be discussed qualitatively. Future ground-water and fish ingestion exposures are some examples. Clearly, the inability to include such exposures in quantitative estimates of risk adds uncertainty to the risk assessment and means that the quantitative estimate of risk from the site is an underestimation. Often, exposure routes that cannot be, or have not been, quantified are expected to make a small contribution to the overall exposure; however, when the opposite is true, further study or monitoring is undertaken in an attempt to quantify the risk.

Exposure Assumptions. Once the concentration of contaminants in water, soil, or air is known by either measurement or modeling, the amount of the contaminants that humans are exposed to must be estimated. This entails making assumptions about human behavior, i.e., how often people are exposed, and what percentage of the contaminant is taken up by those people who are exposed.

Some of the assumptions used to estimate frequency of exposure can be quite uncertain. For example, the frequency at which teenagers visit the Cold Creek/LeMoyne Site and are exposed to chemicals on the site is unknown. Nor is the frequency at which people eat soil known. In the absence of such data, assumptions need to be made to estimate visitation frequency. These depend upon the site's location, surrounding, and accessibility. In the case of the Cold Creek/LeMoyne site, few residences are located near the site and it is located in an industrialized area. Thus, visits by unauthorized people are expected to be infrequent.

Similarly, assumptions need to be made about how much fish, water and soil people ingest and how much of their skin comes into contact with contaminated soil. Many of the assumptions used to estimate these parameters are based on experimental data, so uncertainty is reduced. It is always possible that people visiting a contaminated area have either higher or lower exposures (and risks) than have been assumed. In general, the assumptions used are thought to lead to an overestimate of exposure, rather than an underestimation for most, but not necessarily all, people. Thus, estimates of exposure to contaminants at a site can also make a substantial contribution to the overall uncertainty.

<u>Uptake assumptions</u>. In many cases only a portion of the total amount of a contaminant that a person is exposed to is absorbed. For most compounds that are encountered at a hazardous waste site, including the Cold Creek/LeMoyne site, very little is known about the magnitude of absorption. When such information is available it is used; when it is not, assumptions that are likely to lead to overestimates but are protective of the public health are used, e.g., 100% absorption. Thus a great deal of uncertainty is associated with assumptions about uptake, and the assumptions are more likely to lead to overestimates rather than underestimates of uptake, exposure and risk.

### 7.3 Dose-Response Assessment

Accepted practice divides health effects of concern at hazardous waste sites into two general categories: effects with a threshold, and effects without a threshold.

Dose-response assessments for both of these types of effects share many of the same sources of uncertainty. In the discussion below the more important sources are presented.

Assumptions that are anticipated to create more uncertainty for one class of effects than the other are noted.

Animal to human extrapolation. For many compounds animal studies provide the only reliable information on which to base an estimate of adverse human health effects. Extrapolation from animals to humans introduces a great deal of uncertainty in the risk assessment. Some of this uncertainty can be reduced if the fate of the compound and the mechanism by which it causes adverse effects is known in both animals and humans. When the fate and mechanism is unknown, uncertainty increases. The procedures used to extrapolate from animals to humans make conservative assumptions such that overestimation of effects in humans is far more likely than

underestimation. Nevertheless, because the fate of compounds can differ in humans and animals, it is possible that animal experiments will not reveal an adverse effect that would manifest itself in humans. These can result in an underestimation of the effect in humans. The opposite is also true; effects observed in animals may not be observed in humans, resulting in an overestimation. Thus, animal to human extrapolation can introduce a great deal of uncertainty: an overestimate of the adverse impacts on humans is likely, but an underestimation of the risks cannot be ruled out.

High to low dose extrapolation. The concentration of compounds to which people are exposed at hazardous waste sites is usually much lower than the levels used in the studies from which dose-response relationships are developed. Predicting effects at hazardous waste sites, therefore, requires use of models that allow extrapolation of effects from high These models contain assumptions which to low doses. introduce uncertainty and the uncertainty can be very large. Usually it is larger for carcinogens than for noncarcinogens. As in all the cases discussed above, assumptions are chosen such that overestimation of risk is far more likely than underestimation; however, when the mechanism of action is unknown. there is a possibility that the potential for adverse effects can be underestimated.

### 7.4 Risk Characterization

Based on estimated levels of exposure and dose-response relationships the risk of adverse human health effects is characterized. Two important additional sources of uncertainty are introduced in this phase of the risk assessment: the evaluation of exposure to multiple compounds and the presence of sensitive subpopulations.

- Risk from multiple compounds. Once exposure to and risk from each of the compounds is quantified, the total risk posed by the site is determined by combining the health risk from each of the compounds. Presently, threshold effects are added unless evidence exists indicating that the compounds being investigated interact synergistically or antagonistically with each other. The same is true for carcinogenic effects. For virtually all combinations of compounds at hazardous waste sites, little if any evidence on interaction is available. Therefore, additivity is assumed. The assumption of additivity adds uncertainty and, while the exact magnitude is unknown, it is not expected to be large. Whether assuming additivity leads to an underestimation or overestimation of risk is also unknown and will vary on a case-by-case basis.
- Risk to sensitive populations. The health risks estimated in the risk characterization section generally are applicable to the average resident near the site. Human sensitivity varies from person to person. In some cases it is possible to identify sensitive populations that may be exposed to contaminants on the site and quantify a separate risk for that group. At other times it may not be possible to identify such groups. In all cases, some people will be more sensitive than the average person and, therefore, will be at greater risk. This source of uncertainty is difficult to quantify, but the underestimation of risk due to varying sensitivities is more than compensated for by the use of assumptions throughout the risk assessment that overestimate risk to the average person.

APPENDIX A

DOSE CALCULATION WORK SHEETS

		incidental Soil	Innestron:	Toenagers, Mors	t Case										
Cor			-	# Absorption #		# Duration i	Conversion /	Body weight	= days/year	n lifetime years	Chronic Daily = Intake (mg/kg/day)	Reference Dose (ag/kg/day)	Carcinogenic ( Potency Factor	Horcarcinogenic Risk	Carcinogenic Risk
Carbon Tetrachi Carbon Disc	ulfide	nd	<b>-</b>	•	,.	,	.gg	***		,	ing, ng, coy,	1.006-01	1.30E-01		
	yanıde lercury wates:	nd 690.000	100	1	6	5	0.000001	50	365	70	1.62E-06	5.00E-03		8. 10E-04	
Buty	EPIC late	1,000 1,800	100 100	1	6	5 5	0.000001 0.000001	50 50	365 365 365 365 365	70 70	2.35E-09 4.23E-09	2.50E-02 7.00E-02		9. 39£ -0 <b>0</b> 6. 04£ -0 <b>6</b>	
Versio. Pebu		1, 100 0, 300	100 100	1	6	5 5	0. 000001 0. 000001	50 50	365 365	70 70	2.58E-09 7.05E-10	1.60E-03 1.60E-02		2.58E-06 4.40E-08	
Mola	rate	0. 900	100	į	ě	Š	0.000001	50	365	70	2.11E-09	2.00E-03		1.06E-06	
Cycle Thise	uate yarate	1.800	100	1	•	3	0.000001	50	.365	70	4.2XE-09	5. OOE -03		8. 45£ -07	
		Incidental Soil	ingestion:	Taenagers, Real	istic Case	•					Chronic Baily	Reference	Carcinogenic	Noncarcinogenic	Carcinggenic
Cor Carbon Tetrach	-	Concentration in mg/kg	Owantity Og/visit	x Absorption x	Frequency (15155/yr	# Buration : years	i Conversion i	Body weight kg	m days/year	u lifetime years	= Intake (mg/kg/day)	Dose (ug/kg/day)	Potency Factor 1.30E-01	Ass	Rick
Carbon Dis												1.00E-01 2.00E-02	1.300 01		
M	tercury	45. 731	100	1	6	5	0.000001	50	365	70	1.07E-07	2.00E-03		5. 37E-05	
Thiccarba	EPIC	0, 179	100	1	6	5	0.000001	50	365	70	4. 19E-10	2.50É-02		1.68E-08	
Buty		0.300	100	!	6	5	0.000001	50	365	70 70	7.05E-10 4.36E-10	7, 00E-02 1, 00E-03		1.016-08	
Versic Febu		0.186 0.043	100 100	;	,	5 5	0.000001 0.000001	50 50	365 365 266	70	1.01E-10	1.606-02		4.36E-07 6.30E-09	
	mate	0. 193	100	i	Ĺ	5	0.000001	50	365	70	4.53E-10	2. 60E -03		2.26E-07	
Cyc l Thioc	loate :yarate	0. <b>35</b> 7	100	I		5	0.00001	56.	365	70	<b>a.</b> 39€-10	5. 00E -03		1.68E-07	
		Incidental Soil	Ingestions	On-site Horkers	s, Horst C	15.0								<b>.</b>	
Co		Concent vat con	· Ouzetstu	x Absorption x	France	u Buration :	. Commercian	/ Body seriabt	u dave/war	u lifetime	Chronic Baily  Retake	Merence	Carcinogenic   Potency	Maneare i nogenie lisk	Larcino <b>ge</b> nic Risk
Carbon Tetrach	•	mg/kg	mg/visit		visits/yr	years	kg/mg	ig	,,	years	(mg/kg/day)	(mg/kg/day)	Factor 1,30E-01	***	
Carbon Dis		-										1.00E-01 2.00E-02			
	Cyanide Recours	690, 000	100	1	12	30	0.000001	70	365	70	1.39£-05	2.00E-03		6.94E-03	
Thiocarba		4 ***	100			30	0.000001	70	365	70	2.01E-08	2.50E-02		B. (6E-07	
	EPIC vlate	1.000 1.800	100 100		15	30	0.000001	70	365	70	3.62E-08	7.00E-02		5. 18E -07	
Versio		1.100	100	i	12	30 30	0.000001	70 70	355 355 365 365 365	70	5. SIE-08	1.00E-03		3. 21€- <b>05</b>	
	ulate	0.300	001 001		15	30 30	0.000001 0.000001	70 70	363	70 70	6.04E-09 1.81E-08	1.60E-02 2.00E-03		3.77E-07 9.06E-06	
	inale loate	0.900 1. <b>8</b> 00	100		15	30	0.000001	70	365	70	3.62E-00	5. 00E -03		7.25E-06	
	cyanate	-		·											
			-	On-Site Workers							Chronic Baily	Reference	Carcinogenic	Moncarcinogenic	
Co	copcord	Concentration mg/kg	u Quantity mg/visit	# Absorption =	Frequency visits/yr	x Buration : years	L Conversion : kg/eg	/ Body weight kg	z days/year	n lifetime years	= intake (mg/kg/day)	Dose (mg/kg/day)	Pötency Factor 1.30E-01	Aisk	Azsk
Carbon Tetrach Carbon Dis	sulf ide	nd										1.00E-01 2.00E-02	1. 302 'VI		
	Cyanide Mercury	rd 45. 731	100	1	12	30	0.000001	70	365	70	9. 20E-07	2.00£-03		4.60E-04	
Ihrocarba	mates:		•••	-		•		34	2.5	34.	7 50C 00	3 5/4 A3		1. 44E-07	
	SPIC	0. 179 0. 300	100 100		15	30 30	0.000001 0.000001	70 70	365 365	70 70	1, 59E -09 6, 04E -09	2.50€-02 7.00€-02		1. 442 - 07 8. 63£ - (4)	
	ylate Glate	0.300 0.186	100		15	30	0.000001	70	365 365 365	70	3.746-09	1.00€-03		3.746-06	
	ulate	0.043	100		12	30	0.000001	70	365	70	8. 64E-10	1.606-02		5. 40E -08	
	inate loate	0, 193 0, 357	100 100		15	30 36	0,000001	70 70	<u>¥</u> 5 3€5	70 70	3, 88E -09 7, 19E -09	2,00E-03 5,00E-03		1.94E-(16 1.44E-(16	

	Dermai Hosorpti	on: Teenagers,											<u>.</u> .	
Compound	Concentration mg/kg	Skin area x Contacted cm2	Quantity x on skin ug/cm2	x Absorption	x Frequency visits/yr	x Duration :	Conversion kg/ag	/ Body weight	ж days/year з	lifetime =   years	Chronic Daily Intake (mg/kg/day)	Reference Dose (mg/kg/day)	Carcinogenic Noncarcinoger Potency Ris Factor	
Carbon Tetrachloride Carbon Disulfide	nd		<b></b>	•	,,	,	-99	~•		, co. 2	(mg/ ng/ ou)	1.00E-01	1.30E-01	
Cyanide Mercury Thiocarbanates:	690.000	810	0.5	0	6	5	0.000001	50	365	70	0.00E+00	5.00€-03 5.00€-03	0. 00E+0	)
EPTC	1.000	810	0.5	0.01	6	5	0.000001	50	365	70	9.51E-11	2.506-02	3. 80E →	9
Butylate	1.800	810	0.5	0.01	6	5	0.000001	50	365 365 365	70	1.71E-10	7.00E-02	2.45E-	
Vernolate Pebulate	1.100 0.300	810 810	0.5 0.5	0.01 0.01	, <u>,</u>	5 5	0.000001 0.000001	50 50	.363 365	70 70	1.05E-10 2.85E-11	1.60E-03	1.05E-( 1.78E-(	
Molinate	0.900	810	0.5	0.01	6	5	0.000001	50	365	70	8.56E-11	2.00E-03	4.286-	
Cycloate Thiocyanate	1.800	810	0.5	0.01	6	5	0.000001	50	365	70	1.71E-10	5.00E-03	3. 426-	
	Dermal Absorptio	on: Teenagers,	Realistic C	ase										
		Skin area	Quantity								Chronic Daily	Reference	Carcinogenic Moncarcinogen	ic Carcinogenic
Compound	Concentration			n Absorption				/ Body weight	H days/year :			Dose	Potency Ris	h Risk
Carbon Tetrachloride	mg/kg	cm2	mg/cm2		visits/yr	years	kg/ng	kg		years	(mg/kg/day)	(mg/kg/day)	Factor 1,30E-01	
Carbon Disulfide	-											1.00E-01	1.300	
Cyanide	nd											2.00E-02		
Mercury	45. 731	810	0.5	0	6	5	0.000001	50	365	70	0.00E+00	2.00E-03	0. 00E+	0
Thiocarbanates: EPTC	0.179	810		0.01			A A00001	50	~*	70	1.70E-11	2.50E-02	6.79E-	n
Butylate	0.300	810	0.5 0.5	0. 01 0. 01	6	5	0.000001 0.000001	50	365 365	70 70	2.85E-11	7.00E-02	4.08E-	
Vernolate	0. 186	810	0.5	0.01	ĕ	Š	0.000001	50	365	70	î. 77E-11	1.00E-03	i. 77€ -	
Pebulate	0.043	810	0.5	0.01	Ē	5	0.000001	50 50	365 365 365 365	70	4.08E-12	1.60E-02	2.55E-	
Molinate	0. 193 0. 357	810 810	0.5 0.5	0. 01 0. 01	6	5 5	0.000001 0.000001	50 50	365 365	70 70	1.83E-11 3.40E-11	2.00E-03 5.00E-03	9. 17E⊣ 6. 79E⊣	
Cycloate Thiocyanate	0.337	810	0.5	0.01	ъ	3	0.000001	30	363	70	3. 4VC~11	3. WE-03	6.732~	,
		_		_										
	Dermal Absorption	on: On-site Worl	kers, Worst	Case										
		M.:	O A								Character Badden	9-4	C	in C
Compound	Concentration :			x Absorption	x Frequency			/ Body weight	x days/year :	: lifetime =	Chronic Daily Intake	Dose	Carcinogenic Moncarcinogen Potency Ric	ic Carcinogenic k Risk
•	Concentration a	Skin area r Contacted cm2		и Absorption	x Frequency visits/yr	x Duration :	Conversion kg/ag	/ Body weight	x days/year :	: lifetime = years			Potency Ris Factor	ie Carcinogenie k Risk
Carbon Tetrachloride		r Contacted	# on skin	и Absorption	x Frequency visits/yr			/ Body wright kg	x days/year i	: lifetime = years	Intake	Dose	Potency Ris	ic Carcinogenic k Risk
Carbon Tetrachloride Carbon Disulfide Cyanide	ag/kg nd nd	r Contacted cm2	и on skin sq/cm2	•	visits/yr	years	kg/ag	i kg		years	Intaké (mg/kg/day)	Dose (mg/kg/day) 1.00E-01 2.00E-02	Potency Rid Factor 1, 30E-01	k Azsk
Carbon Tetrachloride Carbon Disulfide Cyanide Mercury		r Contacted	# on skin	и Absorption . O	x Frequency visits/yr			/ Body weight kg	x days/year i	t lifetime = years 70	Intake	Dose (mg/kg/day) 1.00E-01	Potency Ris Factor	k Azsk
Carbon Tetrachloride Carbon Disulfide Cyanide Hercury Thiocarbamates:	mg/kg nd nd 690,000	r Contacted cm2	n on skin mg/cm2	0	visits/yr 6	years 30	kg/sg 0,000001	70	365	years 70	Intaké (mg/kg/day) 0.00E+00	Dose (mg/kg/day) 1.00E-01 2.00E-02 2.00E-03	Potency Ris Factor 1, 30E-01	k RISK O
Carbon Tetrachloride Carbon Disulfide Cyanide Mercury Thiocarbanates: EPTC	ag/kg nd nd	1000 1000 1000	и on skin sq/cm2	•	visits/yr	years 30 30 30	0,000001 0,000001 0,000001	70 70 70 70	365 365	years 70 70 70	Intaké (mg/kg/day) 0.00E+00 5.03E-10 9.06E-10	Dose (mg/kg/day) 1.00E-01 2.00E-02 2.00E-03 2.50E-02 7.00E-02	Potency Ris Factor 1.30E-01 0.00E+0 2.01E-0 1.29E-0	k Risk O B
Carbon Tetrachloride Carbon Disulfide Cyanide Hercury Thiocarbanates: EPTC Butylate Vernolate	#g/kg rd 690.000 1.000 1.800 1.100	1000 1000 1000 1000 1000	u on skin my/cm2 0.5 0.5 0.5 0.5	0 0.01 0.01 0.01	visits/yr	years 30 30 30 30	0,000001 0,000001 0,000001 0,000001	70 70 70 70 70	365 365 365 365	70 70 70 70 70	Intakii (mg/kg/day) 0.00E+00 5.03E+10 9.06E-10 5.54E-10	Dose (mg/kg/day) 1.00E-01 2.00E-02 2.00E-03 2.50E-02 7.00E-02 1.00E-03	Potency Ric Factor 1.30E-01 0.00E+0 2.01E-0 1.29E-0 5.54E-0	k R25k O B B 7
Carbon Tetrachloride Carbon Disulfide Cyanide Hercury Thiocarbanates: EPTC Butylate Vernolate Pebulate	rd 690,000 1.000 1.100 0.300	1000 1000 1000 1000 1000 1000	u on skin mg/cm2 0.5 0.5 0.5 0.5 0.5	0 0.01 0.01 0.01 0.01	visits/yr	30 30 30 30 30 30	0,000001 0,000001 0,000001 0,000001 0,000001	70 70 70 70 70 70	365 365 365 365 365	70 70 70 70 70 70	Intaké (ug/kg/day) 0,00E+00 5,03E-10 9,06E-10 5,54E-10 1,51E-10	Dose (mg/hg/day) 1.00E-01 2.00E-02 2.00E-03 2.50E-02 7.00E-02 1.00E-03 1.60E-02	Pôtemcy Ris Factor 1.30E-01 0.00E+0 2.01E-0 1.29E-0 5.54E-0 9.44E-0	k Risk 0 8 8 7 7
Carbon Tetrachloride Carbon Disulfide Cyanide Mercury Thiocarbamates: EPTC Butylate Vernolate Pebulate Molinate	red 690,000 1,000 1,800 1,100 0,300 0,900	1000 1000 1000 1000 1000 1000 1000	# on skin mg/cm2 0.5 0.5 0.5 0.5 0.5	0.01 0.01 0.01 0.01 0.01	visits/yr	years 30 30 30 30	0,000001 0,000001 0,000001 0,000001 0,000001	70 70 70 70 70	365 365 365 365	70 70 70 70 70	Intaké (mg/kg/day) 0.00E+00 5.03E-10 9.06E-10 5.54E-10 1.51E-10 4.53E-10	Dose (mg/kg/day) 1.00E-01 2.00E-02 2.00E-03 2.50E-02 7.00E-02 1.00E-03 1.60E-02 2.00E-03	Pötency Ris Factor 1.30E-01 0.00E+1 2.01E-1 1.29E-1 5.54E-1 9.44E-2 2.25E-1	k Risk 0 8 8 7 7 9
Carbon Tetrachloride Carbon Disulfide Cyanide Hercury Thiocarbanates: EPTC Butylate Vernolate Pebulate	mg/kg nd 690,000 1.000 1.100 0.300 0.900 1.800	1000 1000 1000 1000 1000 1000	u on skin mg/cm2 0.5 0.5 0.5 0.5 0.5	0 0.01 0.01 0.01 0.01	visits/yr	30 30 30 30 30 30 30	0,000001 0,000001 0,000001 0,000001 0,000001	70 70 70 70 70 70	365 365 365 365 365 365	70 70 70 70 70 70 70	Intaké (ug/kg/day) 0,00E+00 5,03E-10 9,06E-10 5,54E-10 1,51E-10	Dose (mg/hg/day) 1.00E-01 2.00E-02 2.00E-03 2.50E-02 7.00E-02 1.00E-03 1.60E-02	Pôtemcy Ris Factor 1.30E-01 0.00E+0 2.01E-0 1.29E-0 5.54E-0 9.44E-0	k Risk 0 8 8 7 7 9
Carbon Tetrachloride Carbon Disulfide Cyanide Hercury Thiocarbanates: EPTC Butylate Vernolate Pebulate Holinate Cycloate	mg/kg nd 690,000 1.000 1.100 0.300 0.900 1.800	1000 1000 1000 1000 1000 1000 1000 100	# on skin mg/cm2 0.5 0.5 0.5 0.5 0.5 0.5	0.01 0.01 0.01 0.01 0.01 0.01	visits/yr	30 30 30 30 30 30 30	0,000001 0,000001 0,000001 0,000001 0,000001	70 70 70 70 70 70	365 365 365 365 365 365	70 70 70 70 70 70 70	Intaké (mg/kg/day) 0.00E+00 5.03E-10 9.06E-10 5.54E-10 1.51E-10 4.53E-10	Dose (mg/kg/day) 1.00E-01 2.00E-02 2.00E-03 2.50E-02 7.00E-02 1.00E-03 1.60E-02 2.00E-03	Pötency Ris Factor 1.30E-01 0.00E+1 2.01E-1 1.29E-1 5.54E-1 9.44E-2 2.25E-1	k Risk 0 8 8 7 7 9
Carbon Tetrachloride Carbon Disulfide Cyanide Reccury Thiocarbanates: EPTC Sutylate Vernolate Pebulate Molinate Cycloate Thiocyanate	mg/kg nd 690,000 1.000 1.800 1.100 0.300 0.300 1.600	1000 1000 1000 1000 1000 1000 1000 100	# on skin my/cm2 0.5 0.5 0.5 0.5 0.5 0.5 0.5	0 0.01 0.01 0.01 0.01 0.01 0.01	visits/yr 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6	30 30 30 30 30 30 30 30 30	0,000001 0,000001 0,000001 0,000001 0,000001 0,000001	70 70 70 70 70 70 70	365 365 365 365 365 365 365	70 70 70 70 70 70 70 70	Intake (mg/kg/day)  0.00E+00  5.03E-10  9.06E-10  5.54E-10  4.53E-10  9.06E-10	Jose (mg/kg/day) 1.00E-02 2.00E-03 2.50E-02 7.00E-03 1.60E-02 2.00E-03 5.00E-03	Potency Ris Factor 1.30E-01  0.00E+0  2.01E-0 1.29E-0 5.54E-0 9.44E-0 2.25E-0 1.81E-0  Carcinogenic Moncarcinogen	k Risk  0 8 8 7 7 9 7 7
Carbon Tetrachloride Carbon Disulfide Cyanide Reccury Thiocarbanates: EPTC Sutylate Vernolate Pebulate Molinate Cycloate Thiocyanate	mg/kg nd	1000 1000 1000 1000 1000 1000 1000 100	# on skin my/cm2 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5	0 0.01 0.01 0.01 0.01 0.01 0.01	visits/yr 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6	30 30 30 30 30 30 30 30 30 30 30 30 30 3	0,000001 0,000001 0,000001 0,000001 0,000001 0,000001 0,000001	70 70 70 70 70 70 70	365 365 365 365 365 365	70 70 70 70 70 70 70 70 70	Intake (mg/kg/day)  0.00E+00  5.03E-10  9.06E-10  5.5AE-10  1.51E-10  9.06E-10  Chronic Baily Intake	Jose (mg/kg/day) 1.00E-02 2.00E-02 2.00E-03 2.50E-02 1.00E-03 1.60E-02 2.00E-03 5.00E-03	Pôtency Ris Factor 1.30E-01  0.00E+0  2.01E-0 1.29E-0 5.54E-0 9.44E-0 2.20E-1 1.81E-0  Carcinogenic Moncarcinogen Potency Ris	k Risk  0 6 8 7 7 7 7 ic Carcinogenic
Carbon Tetrachloride Carbon Disulfide Cyanide Hercury Thiocarbamates: EPTC Butylate Vernolate Pebulate Molinate Cycloate Thiocyanate	mg/kg nd 690,000 1.000 1.800 1.100 0.300 0.900 1.800 Theory in the content of the	1000 1000 1000 1000 1000 1000 1000 100	# on skin my/cm2 0.5 0.5 0.5 0.5 0.5 0.5 0.5	0 0.01 0.01 0.01 0.01 0.01 0.01	visits/yr 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6	30 30 30 30 30 30 30 30 30	0,000001 0,000001 0,000001 0,000001 0,000001 0,000001	70 70 70 70 70 70 70	365 365 365 365 365 365 365	70 70 70 70 70 70 70 70	Intake (mg/kg/day)  0.00E+00  5.03E-10  9.06E-10  5.54E-10  4.53E-10  9.06E-10	Jose (mg/kg/day) 1.00E-02 2.00E-03 2.50E-02 7.00E-03 1.60E-02 2.00E-03 5.00E-03	Potency Rin Factor 1.30E-01  0.00E+0  2.01E-0 1.29E-0 5.54E-0 9.44E-0 2.26E-0 1.81E-0  Carcinogenic Moncarcinogen Potency Rin Factor	k Risk  0 6 8 7 7 7 7 ic Carcinogenic
Carbon Tetrachloride Carbon Disulfide Cyanide Mercury Thiocarbanates: EPTC Butylate Vernolate Pebulate Molinate Cycloate Thiocyanate  Ccapound Carbon Tetrachloride	mg/kg nd	1000 1000 1000 1000 1000 1000 1000 100	# on skin my/cm2 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5	0 0.01 0.01 0.01 0.01 0.01 0.01	visits/yr 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6	30 30 30 30 30 30 30 30 30 30 30 30 30 3	0,000001 0,000001 0,000001 0,000001 0,000001 0,000001 0,000001	70 70 70 70 70 70 70	365 365 365 365 365 365 365	70 70 70 70 70 70 70 70 70	Intake (mg/kg/day)  0.00E+00  5.03E-10  9.06E-10  5.5AE-10  1.51E-10  9.06E-10  Chronic Baily Intake	Jose (mg/kg/day) 1.00E-02 2.00E-02 2.00E-03 2.50E-02 1.00E-03 1.60E-02 2.00E-03 5.00E-03	Pôtency Ris Factor 1.30E-01  0.00E+0  2.01E-0 1.29E-0 5.54E-0 9.44E-0 2.20E-1 1.81E-0  Carcinogenic Moncarcinogen Potency Ris	k Risk  0 6 8 7 7 7 7 ic Carcinogenic
Carbon Tetrachloride Carbon Disulfide Cyanide Hercury Thiocarbamates: EPTC Butylate Vernolate Pebulate Molinate Cycloate Thiocyanate	mg/kg md 690,000 1.000 1.800 1.100 0.300 0.900 1.800 - Dermal Absorptio	1000 1000 1000 1000 1000 1000 1000 100	# on skin mg/cm2 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5	0 0.01 0.01 0.01 0.01 0.01 tic Case	visits/yr  6 6 6 6 6 6 6 7 x Frequency	30 30 30 30 30 30 30 30 30 30	0,000001 0,000001 0,000001 0,000001 0,000001 0,000001 0,000001	Fig 70 70 70 70 70 70 70 70	365 365 365 365 365 365 365 365	70 70 70 70 70 70 70 70 70	Intake (mg/kg/day) 0,00E+00 5,03E-10 9,06E-10 5,54E-10 4,53E-10 9,06E-10 Chronic Baily Intake (mg/kg/day)	Inse (mg/kg/day) 1.00E-01 2.00E-02 2.00E-03 2.50E-02 7.00E-02 1.00E-03 1.60E-02 2.00E-03 5.00E-03 Reference Dose (mg/kg/day) 1.00E-01 2.00E-01	Potency Factor 1.30E-01 0.00E+0 2.01E-0 1.29E-0 5.54E-0 9.44E-0 2.26E-0 1.81E-0 Carcinogenic Moncarcinogen Potency Factor 1.30E-01	k Risk  8 8 8 7 7 9 9 7 7 7 9 9 7 7 9 9 9 9 9 9
Carbon Tetrachloride Carbon Disulfide Cyanide Mercury Thiocarbanates: EPTC Butylate Vernolate Pebulate Molinate Cycloate Thiocyanate  Ccapound Carbon Tetrachloride Carbon Disulfide Cyanide Mercury	mg/kg nd	1000 1000 1000 1000 1000 1000 1000 100	# on skin my/cm2 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5	0 0.01 0.01 0.01 0.01 0.01 0.01	visits/yr 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6	30 30 30 30 30 30 30 30 30 30 30 30 30 3	0,000001 0,000001 0,000001 0,000001 0,000001 0,000001 0,000001	70 70 70 70 70 70 70	365 365 365 365 365 365 365	70 70 70 70 70 70 70 70 70	Intake (mg/kg/day)  0.00E+00  5.03E-10  9.06E-10  5.5AE-10  1.51E-10  9.06E-10  Chronic Baily Intake	Jose (mg/kg/day) 1.00E-02 2.00E-03 2.50E-02 7.00E-03 1.60E-02 2.00E-03 5.00E-03 Reference Dose (mg/kg/day) 1.00E-01	Potency Rin Factor 1.30E-01  0.00E+0  2.01E-0 1.29E-0 5.54E-0 9.44E-0 2.26E-0 1.81E-0  Carcinogenic Moncarcinogen Potency Rin Factor	k Risk  8 8 8 7 7 9 9 7 7 7 9 9 7 7 9 9 9 9 9 9
Carbon Tetrachloride Carbon Disulfide Cyanide Hercury Thiocarbanates: EPTC Butylate Vernolate Pebulate Holinate Cycloate Thiocyanate  Carbon Tetrachloride Carbon Disulfide Cyanide Hercury Thiocarbanates:	mg/kg rdf rd	1000 1000 1000 1000 1000 1000 1000 100	# on skin mg/cm2 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5	0 0.01 0.01 0.01 0.01 0.01 0.01 0.01 0.	visits/yr  6 6 6 6 6 6 6 7 x Frequency	30 30 30 30 30 30 30 30 30 30 30 30 30 3	0,000001 0,000001 0,000001 0,000001 0,000001 0,000001 0,000001	70 70 70 70 70 70 70 70 70	365 365 365 365 365 365 365 365	70 70 70 70 70 70 70 70 70 70	Intake (mg/kg/day)  0.00E+00  5.03E-10  9.06E-10  5.5AE-10  1.51E-10  4.53E-10  9.06E-10  Chronic Baily Intake (mg/kg/day)  0.00E+00	### ##################################	Potency Ric Factor 1.30E-01  0.00E+0  2.01E-0 1.29E-0 5.54E-0 9.44E-0 2.26E-0 1.81E-0  Carcinogenic Moncarcinogen Potency Ric Factor 1.30E-01  0.00E+0	k Risk  0 8 8 7 7 9 7 7 ic Carcinogenic k Risk
Carbon Tetrachloride Carbon Disulfide Cyanide Hercury Thiocarbamates: EPTC Butylate Vernolate Pebulate Holinate Cycloate Thiocyanate  Carbon Tetrachloride Carbon Disulfide Mercury Thiocarbamates: EPTC	mg/kg nd 690,000 1.000 1.800 1.100 0.300 0.900 1.800 - Dermal Absorption mg/kg nd 45,731 0.179	1000 1000 1000 1000 1000 1000 1000 100	# on skin mg/cm2 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5	0 0.01 0.01 0.01 0.01 0.01 tic Case	visits/yr  6 6 6 6 6 6 7 x Frequency visits/yr	years  30 30 30 30 30 30 30 30 30 30 30 30 30	0,000001 0,000001 0,000001 0,000001 0,000001 0,000001 0,000001	Fig 70 70 70 70 70 70 70 70	365 365 365 365 365 365 365 365	70 70 70 70 70 70 70 70 70	Intake (mg/kg/day)  0.00E+00  5.03E-10  9.06E-10  5.54E-10  9.06E-10  Chronic Baily Intake (mg/kg/day)  0.00E+00  8.99E-11	Ing/kg/day)  1.00E-01 2.00E-02 2.00E-03 2.50E-02 7.00E-03 1.60E-02 2.00E-03 5.00E-03 Reference Dose (mg/kg/day) 1.00E-01 2.00E-02 2.00E-03 2.50E-02	Potency Factor 1.30E-01 0.00E+0 2.01E-0 1.29E-0 5.54E-0 9.44E-0 2.26E-0 1.81E-0 Carcinogenic Moncarcinogen Potency Factor 1.30E-01	k Risk  8 8 8 7 7 9 9 7 7 7 9 9 7 7 9 9 9 9 9 9
Carbon Tetrachloride Carbon Disulfide Cyanide Hercury Thiocarbanates: EPTC Butylate Vernolate Pebulate Holinate Cycloate Thiocyanate  Carbon Tetrachloride Carbon Disulfide Cyanide Hercury Thiocarbanates:	mg/kg rdf rd	1000 1000 1000 1000 1000 1000 1000 100	# on skin mg/cm2 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5	0 0.01 0.01 0.01 0.01 0.01 0.01 0.01 0.	visits/yr  6 6 6 6 6 6 7 x Frequency visits/yr	years  30 30 30 30 30 30 30 30 30 30 30 30 30	0,000001 0,000001 0,000001 0,000001 0,000001 0,000001 0,000001 0,000001 0,000001 0,000001 0,000001 0,000001	70 70 70 70 70 70 70 70 70 70 70 70 70	365 365 365 365 365 365 365 365 365 365	70 70 70 70 70 70 70 70 70 70 70 70	Intake (mg/kg/day)  0.00E+00  5.03E-10  9.06E-10  5.5AE-10  1.51E-10  9.06E-10  Chronic Baily Intake (mg/kg/day)  0.00E+00  8.98E-11  1.51E-10  9.34E-11	Mose (mg/kg/day) 1.00E-02 2.00E-03 2.50E-02 7.00E-03 1.60E-02 2.00E-03 5.00E-03 6reference Dose (mg/kg/day) 1.00E-01 2.50E-02 7.00E-02 1.00E-03	Potency Rin Factor 1.30E-01  0.00E+0  2.01E-0 1.29E-0 5.54E-0 9.44E-0 2.26E-0 1.81E-0  Carcinogenic Moncarcinogen Potency Rin Factor 1.30E-01  0.00E+0 3.59E-0 2.16E-0 9.34E-0 9.34E-0	k Risk  0 8 8 7 7 9 7 7 ic Carcinogenic k Risk
Carbon Tetrachloride Carbon Disulfide Cyanide Hercury Thiocarbanates: EPIC Butylate Vernolate Pebulate Holinate Cycloate Thiocyanate  Carbon Tetrachloride Carbon Disulfide Cyanide Mercury Thiocarbanates: EPIC Butylate Vernolate Pebulate	mg/kg nd	1000 1000 1000 1000 1000 1000 1000 100	# on skin mg/cm2 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5	0 0.01 0.01 0.01 0.01 0.01 0.01 0.01 0.	x Frequency visits/yr	years  30 30 30 30 30 30 30 30 30 30 30 30 30	0,000001 0,00001 0,00001 0,00001 0,00001 0,00001 0,00001 0,00001 0,00001 0,00001 0,00001 0,00001 0,00001 0,00001	Fig. 70 70 70 70 70 70 70 70 70 70 70 70 70	365 365 365 365 365 365 365 365 365 365	70 70 70 70 70 70 70 70 70 70 70 70 70 7	Intake (mg/kg/day)  0.00E+00  5.03E-10  9.06E-10  5.54E-10  9.06E-10  Chronic Baily Intake (mg/kg/day)  0.00E+00  8.99E-11  1.51E-10  9.34E-11  2.16E-11	Reference (mg/kg/day) 1.00E-01 2.00E-02 2.00E-03 2.50E-02 7.00E-02 2.00E-03 5.00E-03 Reference (mg/kg/day) 1.00E-01 2.00E-02 2.00E-02 2.00E-03 1.50E-02 1.00E-03	Potency Factor 1.30E-01 0.00E+0 2.01E-0 1.29E-0 5.54E-0 9.44E-0 2.26E-0 1.81E-0 Carcinogenic Moncarcinogen Potency Factor 1.30E-01 0.00E+0 3.59E-0 2.16E-0 9.34E-0 1.35E-0 1.3	k Risk  8 8 8 7 7 9 9 7 7 7 9 9 7 7 9 9 9 9 9 9
Carbon Tetrachloride Carbon Disulfide Cyanide Mercury Thiocarbanates: EPTC Butylate Vernolate Pebulate Molinate Cycloate Thiocyanate  Ccapound Carbon Tetrachloride Carbon Disulfide Cyanide Mercury Thiocarbanates: EPTC Butylate Vernolate Pebulate Pebulate Pebulate Molinate Rolinate	mg/kg rdd	1000 1000 1000 1000 1000 1000 1000 100	# on skin mg/cm2 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5	0 0.01 0.01 0.01 0.01 0.01 0.01 0.01 0.	x Frequency visits/yr	years  30 30 30 30 30 30 30 30 30 30 30 30 30	0,000001 0,000001 0,000001 0,000001 0,000001 0,000001 0,000001 0,000001 0,000001 0,000001 0,000001 0,000001 0,000001 0,000001	Fig. 70 70 70 70 70 70 70 70 70 70 70 70 70	365 365 365 365 365 365 365 365 365 365	70 70 70 70 70 70 70 70 70 70 70 70 70 7	Intake (mg/kg/day)  0.00E+00  5.03E-10  9.06E-10  5.54E-10  9.06E-10  Chronic Baily Intake (mg/kg/day)  0.00E+00  8.99E-11  1.51E-10  9.34E-11  2.16E-11  9.71E-11	Ing/kg/day) 1.00E-02 2.00E-02 2.00E-03 2.50E-02 7.00E-03 1.60E-03 1.60E-03 2.50E-03 6ag/kg/day) 1.00E-03 2.50E-02 2.00E-03 1.50E-02 2.00E-03 1.50E-02 2.00E-03	Potency Ris Factor 1.30E-01  0.00E+0  2.01E-0 1.29E-0 5.54E-0 9.44E-0 2.26E-0 1.81E-0  Carcinogenic Moncarcinogen Potency Factor 1.30E-01  0.00E+0 3.59E-0 9.34E-0 1.35E-0 4.85E-0 4.85E-0 4.85E-0	k Risk  B B B B B B B B B B B B B B B B B B B
Carbon Tetrachloride Carbon Disulfide Cyanide Hercury Thiocarbanates: EPIC Butylate Vernolate Pebulate Holinate Cycloate Thiocyanate  Carbon Tetrachloride Carbon Disulfide Cyanide Mercury Thiocarbanates: EPIC Butylate Vernolate Pebulate	mg/kg rdd 690.000 1.000 1.800 1.100 0.300 0.900 1.800 1.800  Concentration mg/kg rdd 45.731 0.179 0.300 0.186 0.043 0.193 0.357	1000 1000 1000 1000 1000 1000 1000 100	# on skin mg/cm2 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5 0.5	0 0.01 0.01 0.01 0.01 0.01 0.01 0.01 0.	x Frequency visits/yr	years  30 30 30 30 30 30 30 30 30 30 30 30 30	0,000001 0,00001 0,00001 0,00001 0,00001 0,00001 0,00001 0,00001 0,00001 0,00001 0,00001 0,00001 0,00001 0,00001	Fig. 70 70 70 70 70 70 70 70 70 70 70 70 70	365 365 365 365 365 365 365 365 365 365	70 70 70 70 70 70 70 70 70 70 70 70 70 7	Intake (mg/kg/day)  0.00E+00  5.03E-10  9.06E-10  5.54E-10  9.06E-10  Chronic Baily Intake (mg/kg/day)  0.00E+00  8.99E-11  1.51E-10  9.34E-11  2.16E-11	Reference (mg/kg/day) 1.00E-01 2.00E-02 2.00E-03 2.50E-02 7.00E-02 2.00E-03 5.00E-03 Reference (mg/kg/day) 1.00E-01 2.00E-02 2.00E-02 2.00E-03 1.50E-02 1.00E-03	Potency Factor 1.30E-01 0.00E+0 2.01E-0 1.29E-0 5.54E-0 9.44E-0 2.26E-0 1.81E-0 Carcinogenic Moncarcinogen Potency Factor 1.30E-01 0.00E+0 3.59E-0 2.16E-0 9.34E-0 1.35E-0 1.3	k Risk  B B B B B B B B B B B B B B B B B B B

	Ingestion of Co				_			Chronic Daily	Reference	Carcinogenic N Potency	loncard inogenio	Carcinogeni
Compound	Concentration mg/kg	x Quaritity mg/day	n Absorption i	Duration years	x Conversion . kg/mg	/ Body weight kg	x lifetime years	= Intaké (mg/kg/day)	Dose (mg/kg/day)	Potency Factor	Aisk	Ass
a bon letrachloride Earbon Disulfide										1.30E-01		
Cyanide									1.00E-01			
Hercury	3, 100	26000	1	30	0.000001	70	70	4.93E-04	2.00E-02 2.00E-03		2.47E-01	
Threcarbanates:			•	•	***************************************	,,		1. AL VI	E. VOE '03		E. 4/E-01	
EPIC	-								2.506-02			
Butylate	-								7.00E-02		•	
Vernolate	-								1.00E-03			
Pebulate Molinate	-								1.60E-02			
Cycloate	_								2.00E-03 5.00E-03			
Thiocyanate	-								3.002 03			
Adults:	Ingestion of Co	ontaminated F	ish: Nealisti	c Case				Church Brile		C		
Compound	Concentration	u Duankitu	a Observation a	Duration	u Conversion	/ Body smisht	. lifetim	Chronic Daily = Intake	Dose	Carcinogenic N Potency	Dnearcinogenic Risk	Right
	ag/kg	eg/day	- 1222 y 1 1 4 1 1	years	kg/mg	kg	years	(mg/kg/day)	(mg/kg/day)	Factor		71.
rbon Tetrachionide	-									1.30E-01		
Carbon Disulfide	-								1.00E-01			
Cyanide									2.00E-02			
Mercury	1.232	56000	1	30	0.000001	70	70	1.96E-04	2.00E-03		9. BIE-02	
Throcarbamates: EPTC									2.50E-02			
Putylate	-								7,00E-02			
Vernolate	-								1.00E-03			
Pebulate	-								1,60€-02			
Molinate	•								2.00E-03			
Cycloate	-								5. 00E-03			
Thiocyanate	Ingestion of Co	ontaminated F	igh: Norst Ca	se			`		5, 00E -03			
Thiocyanate Teenagers:					n Conversion : kg/mg	f Body weight lig		Chronic Daily	5, 00E-03	Carcinogenic M Polancy Factor	loncarcinogenic Risk	Carcinogen Ris
Thiocyanate  Teenagers:  Ecopound  bon Tetrachtoride	Ingestion of Co Concentration mg/kg	n Quantity		<b>Durat</b> ion			» lifetime	Chronic Baily Intake	5, 00E-03 Reference Bose (ug/kg/day)	Potency	loncarcinogenic Risk	Carcinogen Ris
Thiocyanate  Teenagers;  Ecopound  bon Tetrachtoride  Carbon Disulfide	Ingestion of Co Concentration ag/kg	n Quantity		<b>Durat</b> ion			» lifetime	Chronic Baily Intake	5,00E-03 Reference Bose (ug/kg/day) 1,00E-01	Potency Factor	bnearcinograic Rish	Carcinogeni Ris
Thiocyanate Teenagers; Compound rbon Tetrachtoride Carbon Disulfide Cyanide	Ingestion of Co Concentration mg/kg	n Deantity mg/day	n Misorption n	Buration years	kg/mg	· ƙg	= lifetime years	Chronic Baily * Intake (sg/kg/day)	S, 00E-03 Reference Bose (ug/kg/day) 1.00E-01 2.00E-02	Potency Factor	flish	Carcinogen Ris
Thiocyanate Teenagers; Compound rbon Tetrachtoride Carbon Disulfide Cyanide Mercury	Ingestion of Co Concentration ag/kg	n Deantity mg/day		<b>Durat</b> ion			» lifetime	Chronic Baily Intake	5,00E-03 Reference Bose (ug/kg/day) 1,00E-01	Potency Factor	loncarcinogenic Risk S. 76E-02	Carcinogen Ris
Thiocyanate Teenagers; Compound ton Tetrachiorsid Carbon Bisselfside Eyanide Rencury Thiocarbinates;	Ingestion of Co Concentration mg/kg	n Deantity mg/day	n Misorption n	Buration years	kg/mg	· ƙg	= lifetime years	Chronic Baily * Intake (sg/kg/day)	5, 00E-03 Reference Bose (ug/kg/day) 1,00E-01 2,00E-02 2,00E-02	Potency Factor	flish	Carcinogen Ri
Thiocyanate Teenagers: Compound then Tetrachioride Carbon Disulfide Cyanide Executy Thiccarbanates: EPIC Butylate	Ingestion of Co Concentration mg/kg	n Deantity mg/day	n Misorption n	Buration years	kg/mg	· ƙg	= lifetime years	Chronic Baily * Intake (sg/kg/day)	5.00E-03 Reference Bose (ug/kg/day) 1.00E-01 2.00E-02 2.00E-03 2.50E-02 7.00E-02	Potency Factor	flish	Carcinogen Ri
Thiocyanate Teenagers: Compound rbon Tetrachioride Carbon Dissifide Eyanide Recoury Thiocarbamates: EPIC Butylate Vernolate	Ingestion of Co Concentration mg/kg - - 3, 100	n Deantity mg/day	n Misorption n	Buration years	kg/mg	· ƙg	= lifetime years	Chronic Baily * Intake (sg/kg/day)	5. 00E-03 Reference Bose (eg/kg/day) 1. 00E-01 2. 00E-02 2. 00E-02 7. 00E-02 1. 00E-03	Potency Factor	flish	Carcinogen Rs
Thiocyanate Teenagers: Compound rbon Tetrachtoride Carbon Disulfide Recury Thiocarbanates: EPIC Butylate Vernolate Prebulate	Ingestion of Co Concentration mg/kg - - 3, 100	n Deantity mg/day	n Misorption n	Buration years	kg/mg	· ƙg	= lifetime years	Chronic Baily * Intake (sg/kg/day)	5, 00E-03 Reference Bose (ug/kg/day) 1, 00E-01 2, 00E-02 2, 00E-03 2, 50E-02 7, 00E-03 1, 60E-02	Potency Factor	flish	Carcinogen Ri
Thiocyanate Teenagers: Compound rbon Tetrachioride Carbon Disulfide Eyanide Reccury Thiccarbamates: EPIC Butylate Vernolate Pebulate Holinate	Ingestion of Co Concentration mg/kg 	n Deantity mg/day	n Misorption n	Buration years	kg/mg	· ƙg	= lifetime years	Chronic Baily * Intake (sg/kg/day)	5. 00E-03 Reference Bose (mg/kg/day) 1. 00E-01 2. 00E-02 2. 00E-03 2. 50E-02 1. 00E-03 1. 60E-02 2. 00E-03	Potency Factor	flish	Carcinogen Ris
Thiocyanate Teenagers: Compound rbon Tetrachtoride Carbon Disulfide Recury Thiocarbanates: EPIC Butylate Vernolate Prebulate	Ingestion of Co Concentration mg/kg 	n Deantity mg/day	n Misorption n	Buration years	kg/mg	· ƙg	= lifetime years	Chronic Baily * Intake (sg/kg/day)	5, 00E-03 Reference Bose (ug/kg/day) 1, 00E-01 2, 00E-02 2, 00E-03 2, 50E-02 7, 00E-03 1, 60E-02	Potency Factor	flish	Carcinogeni Ris
Thiocyanate Teenagers: Compound rbon Tetrachtoride Carbon Bisulfide Recoury Thircarbamates: EPTC Butylate Vernolate Pebulate Holimate Cycloate Thiocyanate	Ingestion of Co Concentration mg/kg 	n Quantity mg/day 26000	n Misorption n	Buration years 5	kg/mg	· ƙg	= lifetime years	Chronic Baily Intake (mg/kg/day) 1.15E-04	5. 00E-03 Reference Bose (ug/kg/day) 1. 00E-01 2. 00E-02 2. 00E-03 1. 60E-02 1. 00E-03 5. 00E-03	Pôtency Factor 1.30E-01	Aisk 5. 76E-02	Au
Thiocyanate Teenagers: Compound rbon Tetrachtoride Carbon Bisulfide Eyanide Reccury Thiccarbanates: EPIC Butylate Vernolate Pebulate (Cycloate Thiocyanate Teenagers:	Ingestion of Co Concentration mg/kg - - 3, 100 - - - - -	n Deantity mg/day 26000	n Musorption n i ish: Mealisti	Buration years 5	kg/mg	- Kg 50	≥ lifetium years 70	Chronic Baily  Intake (mg/kg/day)  1.15E-04	5. 00E-03 Reference Bose (ug/kg/day) 1. 00E-01 2. 00E-02 2. 00E-03 1. 60E-02 1. 00E-03 5. 00E-03	Potency Factor	Aisk 5. 76E-02	Ris
Thiocyanate Teenagers: Compound Thioc Tetrachior side Carbon Disself side Eyanide Recrumy Thiocarbanates: EPIC Butylate Vernolate Pubulate Eycloate Thiocyanate Teenagers: Compound	Ingestion of Co Concentration mg/kg - - 3,100 - - - - - - - - - - - - - - - - - - -	n Duantity mg/day 26000 ontaminated F	n Musorption n i ish: Mealisti	Puration years  5 c Case Duration	<b>hg/mg</b> 0,000001  1 Conversion	fig 50	<pre>» lifetime years  70  x Lifetime</pre>	Chronic Baily intake (mg/kg/day)  1.15E-04  Dironic Baily = Intake	5. 00E-03 Reference Bose (ug/kg/day) 1. 00E-01 2. 00E-02 2. 00E-02 7. 00E-02 1. 00E-03 1. 60E-02 2. 00E-03 5. 00E-03	Pôtency Factor 1-30E-01 Carcinogenic M Potency	S. 76E-02	This This Table 1
Thiocyanate Teenagers: Compound Thioc Tetrachior side Carbon Disself side Eyanide Recrumy Thiocarbanates: EPIC Butylate Vernolate Pubulate Eycloate Thiocyanate Teenagers: Compound	Ingestion of Co Concentration mg/kg 3,100	n Duantity mg/day 26000 ontaminated F	n Musorption n i ish: Mealisti	Puration years  5 c Case Duration	<b>hg/mg</b> 0,000001  1 Conversion	fig 50	<pre>» lifetime years  70  x Lifetime</pre>	Chronic Baily intake (mg/kg/day)  1.15E-04  Dironic Baily = Intake	5. 00E-03 Reference Bose (eg/kg/day) 1. 00E-01 2. 00E-02 2. 00E-03 2. 50E-03 1. 60E-02 2. 00E-03 5. 00E-03	Pôtency Factor 1.30E-01 Carcinogenic N Potency Factor	S. 76E-02	Th:
Thiocyanate Teenagers:	Ingestion of Co Concentration mg/kg 	n Duantity mg/day 26000  ontaminated F m Duantity mg/day	n Musorption n i ish: Realisti x Absorption x	Buration years 5 c Case Duration years	o, occors  Conversion kg/mg	fig 50 / Body weight fig	m lifetime years 70 K lifetime years	Chronic Baily intake (mg/kg/day)  1.15E-04  Dironic Baily telake (mg/kg/day)	5, 00E-03  Reference Bose (ug/kg/day)  1, 00E-01 2, 00E-02 2, 00E-03 1, 60E-02 2, 00E-03 5, 00E-03  Reference Bose (ug/kg/day)  1, 00E-01 2, 00E-01	Pôtency Factor 1.30E-01 Carcinogenic N Potency Factor	fish 5.76E-02 boncarcinogenic fish	Th:
Thiocyanate Teenagers:	Ingestion of Co Concentration mg/kg 3,100 	n Duantity mg/day 26000 ontaminated F	n Musorption n i ish: Mealisti	Puration years  5 c Case Duration	<b>hg/mg</b> 0,000001  1 Conversion	fig 50	<pre>» lifetime years  70  x Lifetime</pre>	Chronic Baily intake (mg/kg/day)  1.15E-04  Dironic Baily = Intake	5. 00E-03 Reference Bose (eg/kg/day) 1. 00E-01 2. 00E-02 2. 00E-03 2. 50E-03 1. 60E-02 2. 00E-03 5. 00E-03	Pôtency Factor 1.30E-01 Carcinogenic N Potency Factor	S. 76E-02	This This Table 1
Thiocyanate Teenagers:	Ingestion of Co Concentration mg/kg 	n Duantity mg/day 26000  ontaminated F m Duantity mg/day	n Musorption n i ish: Realisti x Absorption x	Buration years 5 c Case Duration years	o, occors  Conversion kg/mg	fig 50 / Body weight fig	m lifetime years 70 K lifetime years	Chronic Baily intake (mg/kg/day)  1.15E-04  Dironic Baily telake (mg/kg/day)	5. 00E-03 Reference Bose (mg/kg/day) 1. 00E-01 2. 00E-02 2. 00E-03 1. 60E-02 2. 00E-03 5. 00E-03 Reference Bose (mg/kg/day) 1. 00E-01 2. 00E-02 2. 00E-03	Pôtency Factor 1.30E-01 Carcinogenic N Potency Factor	fish 5.76E-02 boncarcinogenic fish	Th:
Thiocyanate Teenagers: Compound rbon Tetrachtoride Carbon Bisulfide Recoury Thircarbamates: EPTC Butylate Vernolate Pebulate Hilinate Cycloate Thiocyanate Teenagers: Compound rbon Tetrachloride Carbon Disulfide Cyanide Hercury Thiocarbamates: EPTC	Ingestion of Co Concentration mg/kg 	n Duantity mg/day 26000  ontaminated F m Duantity mg/day	n Musorption n i ish: Realisti x Absorption x	Buration years 5 c Case Duration years	o, occors  Conversion kg/mg	fig 50 / Body weight fig	m lifetime years 70 K lifetime years	Chronic Baily intake (mg/kg/day)  1.15E-04  Dironic Baily telake (mg/kg/day)	5, 00E-03 Reference Bose (ug/kg/day) 1, 00E-01 2, 00E-02 2, 00E-03 3, 00E-03 5, 00E-03 Reference lose (ug/kg/day) 1, 00E-01 2, 00E-02 2, 00E-03 3, 00E-03	Pôtency Factor 1.30E-01 Carcinogenic N Potency Factor	fish 5.76E-02 boncarcinogenic fish	Th:
Thiocyanate Teenagers:	Ingestion of Co Concentration mg/kg 	n Duantity mg/day 26000  ontaminated F m Duantity mg/day	n Musorption n i ish: Realisti x Absorption x	Buration years 5 c Case Duration years	o, occors  Conversion kg/mg	fig 50 / Body weight fig	m lifetime years 70 K lifetime years	Chronic Baily intake (mg/kg/day)  1.15E-04  Dironic Baily telake (mg/kg/day)	5. 00E-03 Reference Bose (ug/kg/day) 1. 00E-01 2. 00E-02 2. 00E-03 1. 60E-02 2. 00E-03 5. 00E-03 1. 00E-03 2. 00E-03 2. 00E-03 2. 00E-03 2. 00E-03 2. 00E-03	Pôtency Factor 1.30E-01 Carcinogenic N Potency Factor	fish 5.76E-02 boncarcinogenic fish	Th:
Thiocyanate  Teenagers:	Ingestion of Co Concentration mg/kg 	n Duantity mg/day 26000  ontaminated F m Duantity mg/day	n Musorption n i ish: Realisti x Absorption x	Buration years 5 c Case Duration years	o, occors  Conversion kg/mg	fig 50 / Body weight fig	m lifetime years 70 K lifetime years	Chronic Baily intake (mg/kg/day)  1.15E-04  Dironic Baily telake (mg/kg/day)	5, 00E-03 Reference Bose (ug/kg/day) 1, 00E-01 2, 00E-02 2, 00E-03 3, 00E-03 5, 00E-03 Reference lose (ug/kg/day) 1, 00E-01 2, 00E-02 2, 00E-03 3, 00E-03	Pôtency Factor 1.30E-01 Carcinogenic N Potency Factor	fish 5.76E-02 boncarcinogenic fish	Th:
Thiocyanate  Teenagers:	Ingestion of Co Concentration mg/kg 	n Duantity mg/day 26000  ontaminated F m Duantity mg/day	n Musorption n i ish: Realisti x Absorption x	Buration years 5 c Case Duration years	o, occors  Conversion kg/mg	fig 50 / Body weight fig	m lifetime years 70 K lifetime years	Chronic Baily intake (mg/kg/day)  1.15E-04  Dironic Baily telake (mg/kg/day)	5. 00E-03 Reference Bose (mg/kg/day) 1. 00E-01 2. 00E-02 2. 00E-03 1. 60E-02 2. 00E-03 5. 00E-03 1. 60E-02 2. 00E-03 2. 00E-03 2. 00E-02 2. 00E-03 2. 50E-02 2. 00E-03	Pôtency Factor 1.30E-01 Carcinogenic N Potency Factor	fish 5.76E-02 boncarcinogenic fish	Ris
Thiocyanate  Teenagers: Compound  rbon Tetrachtoride Carbon Bisulfide Eyanide Recury Thircarbamates: EPIC Butylate Vernolate Prebulate Hilinate Cycloate Thiocyanate Teenagers: Compound  rbon Tetrachloride Cyanide Microbamates: EPIC Butylate Vernolate Precury Thiocarbamates: EPIC Butylate Vernolate Pebulate Pebulate Pebulate	Ingestion of Co Concentration mg/kg 	n Duantity mg/day 26000  ontaminated F m Duantity mg/day	n Musorption n i ish: Realisti x Absorption x	Buration years 5 c Case Duration years	o, occors  Conversion kg/mg	fig 50 / Body weight fig	m lifetime years 70 K lifetime years	Chronic Baily intake (mg/kg/day)  1.15E-04  Dironic Baily telake (mg/kg/day)	5. 00E-03  Reference Bose (ug/kg/day)  1. 00E-01 2. 00E-02 2. 00E-02 2. 00E-03 3. 00E-03 5. 00E-03  Reference (ug/kg/day)  1. 00E-01 2. 00E-02 2. 00E-03 3. 00E-03 3. 00E-03 1. 60E-01 2. 00E-02 2. 00E-03 3. 00E-03 3. 60E-02	Pôtency Factor 1.30E-01 Carcinogenic N Potency Factor	fish 5.76E-02 boncarcinogenic fish	Ris

Compound	Ingestion of (	Contaminated E	iroundwater fi	rca Downgrad	ient Wells: M	orst Case					
Cartan tak	f Concentration mg/l	x Quantity 1/day	* Absorption	<pre># Duration ; years</pre>	Body weight i	lifetime = years	Chronic Daily Intake (mg/kg/day)	Reference Bose	Carcinogenic Potenc	Moncarcinogenic	Carcinogenic
Carbon Tetrachloride	0.225	2			_	,	.mg/ ng/ ugy/	(mg/kg/day)	Facto	, nisk	Risk
Carbon Disulfide Cyanide	,	5	i	30 30	70 70	70 70	2. 75E -03 5. 56E -04	1,00E-01	1. <b>30E</b> -0	1	3.57E-04
Hercury Thiocarbanates:							J. 332 VV	2.00E-02 2.00E-03		5. 56E -03	3. 3/E -Q4
EPIC	0.021	5	1	34				E. VOE -UJ			
Butylate Versolate	0.007	5 5	i	30	70	70	2.57E-04	2.50E-02			
Pebulate Pebulate	0.008	ž	i	30 30 30	70	70	8. 33E-05	7.00E-02		1.0X-02	
Molinate			•	30	70	70	9. IBE-05	1.00E-03		I. 19E-03	
Cycloate	0.026	5	1	20	7.0			1.60E-02		9. IEE-02	
	0.006	2	i	30 30	70	70	3. IEE-04	2.006-03			
Thiocyanate			•	<b></b>	70	70	6. 98E-05	5. 00E-03		1.59E-01 1.40E-02	
Compound (	Ingestion of Concentration	ntaminated Gr	oundnaterin E Abscration =	bungradient	Mells: Realis	tic Case	Chronic Baily	Marana	Campinana		
compound (	Concentration   mg/l	R Quantity H 1/day	oundusterin B Absorption a	bingradient Buration / years	Hells: Realis Body weight H	tic Case lifetime = years	Chronic Baily Intake (mg/kg/day)		rotenty	Moncarcinogonic Nish	Cercinogenic Risk
Carbon Tetrachloride	Concentration   mg/l 0.0332	R Quantity H 1/day	oundusterin B Absorption s	Buration / years	Body weight a lig	lifetime = years	Intaké (mg/kg/day)	Reference Bose (ug/kg/day)	Carcinogenic Potency Factor	Moncarcinogenic Nish	Carcinogenic Risk
Carbon Tetrachloride Carbon Disulfide Cyanide	Concentration   mg/l	x Owantity H	Oundusterin B Absorption x I I	Peration /	Body weight u	lifetime =	Intaké (mg/kg/day) 4.07E-04	(mg/kg/day)	rotenty	Mish	Carcinogenic Risk 5,28E-05
Carbon Tetrachloride Carbon Diswlfide Cyanide Mercury	Concentration   mg/l 0.0332	R Quantity H 1/day	oundusterin B Absorption s I I	Buration / years	Body weight u kg 70	lifetime = years 70	Intaké (mg/kg/day)	(ag/kg/day) 1.00E-01 2.00E-02	Factor	Noncarcinogenic lisk 3.67E-04	fisk
Carbon Tetrachloride Carbon Disulfide Cyanide	0.0332 0.0030	M Owantity II 1/day 2 2	oundwaterin B Absorption x	Peration / years 30 30	Body weight u kg 70	lifetime = years 70	Intaké (mg/kg/day) 4.07E-04	(mg/kg/day)	Factor	Mish	fisk
Carbon Tetrachloride Carbon Diswifide Cyanide Hercury Thiocarbamates:	0.0332 0.0030 0.0016	M Owantity II 1/day 2 2	oundnater in B Absorption =	Peration / years 30 30	Body weight u kg 70	lifetime = years 70 70	Intaké (ng/kg/day) 4.07E-04 3.67E-05	1.00E-01 2.00E-02 2.00E-03	Factor	Mish	fisk
Carbon Tetrachloride Carbon Disulfide Cyanide Rercury Thiocarbonates: EPTC	0.0332 0.0030 0.0016 0.0005	* Ovantity   1 1/day   2 2 2	conductorin B Absorption # ! !	######################################	Body weight u kg 70 70 70 70	lifetime = years 70 70 70	Int até (ng/kg/day) 4.07E-04 3.67E-05	1.00E-01 2.00E-02 2.00E-03 2.50E-02	Factor	3.67E-04	fisk
Carbon Tetrachloride Carbon Disulfide Cyanide Mercury Thiocarbanates: EPTC Butylate Vernolate Febulate	0.0332 0.0030 0.0016	M Owantity II 1/day 2 2	oundester in 8 Absorption =  1 1 1 1	Peration / years 30 30	Body weight H fig 70 70 70	1 i fet ime = years 70 70 70 70 70 70 70	Int aké (mg/kg/day) 4.07E-04 3.67E-05 1.96E-05 6.12E-06	1.00E-01 2.00E-02 2.00E-03 2.50E-02 7.00E-02	Factor	Mish	fisk
Carbon Tetrachloride Carbon Disulfide Cyanide Rercury Thiocarbanates: EPTC Butylate Vernolate Pebulate Rolinate	0.0332 0.0030 0.0016 0.0005	* Grantity # 1/day   2   2   2   2   2   2   2   2   2	Conductorin B Absorption x  1 1 1	Buration / years 30 30 30 30 30 30 30 30 30 30 30 30 30	Rody weight u tug 70 70 70 70 70 70	lifetime = years 70 70 70	Int até (ng/kg/day) 4.07E-04 3.67E-05	1.00E-01 2.00E-02 2.00E-03 2.50E-02 7.00E-02 1.00E-03	Factor	3. 67E-04-7. 84E-04	fisk
Carbon Tetrachloride Carbon Disulfide Cyanide Rercury Ihiocarbamates: EPTC Butylate Vernolate Pebulate Holinate Cycloate	0.0332 0.0030 0.0030 0.0016 0.0005 0.0005	* Grantity # 1/day   2   2   2   2   2   2   2   2   2	Conductor in B Absorption #  i i i i i	Buration / years 30 30 30 30 30 30 30 30 30 30 30 30 30	Body weight u kg 70 70 70 70 70 70 70	1 i fet ime = years 70 70 70 70 70 70 70 70 70 70 70 70 70	Int aké (mg/kg/day) 4.07E-04 1.67E-05 1.96E-05 6.12E-06 6.12E-06	1.00E-01 2.00E-02 2.00E-03 2.50E-02 7.00E-02 1.00E-03 1.60E-02	Factor	7.84E-04 8.75E-05	fisk
Carbon Tetrachloride Carbon Disulfide Cyanide Rercury Thiocarbanates: EPTC Butylate Vernolate Pebulate Rolinate	0.0332 0.0030 0.0016 0.0005	* Ovantity   1 1/day   2 2 2	oundester in 8 Absorption x  1 1 1 1 1 1	######################################	Rody weight u tug 70 70 70 70 70 70	1 i fet ime = years 70 70 70 70 70 70 70	Int aké (mg/kg/day) 4.07E-04 3.67E-05 1.96E-05 6.12E-06	1.00E-01 2.00E-02 2.00E-03 2.50E-02 7.00E-02 1.00E-03	Factor	7.84E-04 8.75E-05	fisk

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